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ORIGINAL ARTICLES.

AN EFFORT TO OBTAIN A PERFECT SUBSTITUTE FOR HUMAN MILK.

Based on Chemical and Clinical Studies.[1]

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THERE is no more important subject in obstetric practice to-day than the artificial alimentation of infants. Indeed, there are few questions in the whole realm of medicine that transcend it in interest. In this city, in the past three years, 1887 to 1889 inclusive, there have been, in round numbers, 75,000 living children born; during the same period 16,000 children have died before completing their first year—a death-rate of something over 21 per cent.[2] In other words, one-fifth of the children born alive in Philadelphia die before they are one year old—a stupendous mortality, but other large cities show a still worse record. The chief cause of the high death-rate is undoubtedly to be found in diseases of the digestive tract, which come from bad feeding. This does not appear so plainly as it should in the mortality records of the city. One needs little experience to learn that fatal cases of intestinal infection in young children are reported in the death-certificates under all sorts of misleading names, such as, marasmus, cerebritis, convulsions, inanition, debility, etc. But the fact remains indisputable, that if digestive disturbances could be eliminated from the fatal disorders of infancy, a vast army of children would be saved to their parents, and a large number of citizens be added to the State, with a gain, from a humanitarian point of view, that is incalculable, and from a purely material point of view that could be reckoned in enormous sums of money.

By the march of modern investigation such a result, in great part at least, has become a possibility. It needs only a wide dissemination among the laity and the medical profession of knowledge now in the possession of, comparatively, a few, greatly to reduce the mortality of early infancy. That a large number of physicians need instruction on this subject cannot be denied. Last summer I saw two striking illustrations of the fact. In one case, a child of three months was fed, by order of its physician, on a mixture of one teaspoonful of condensed milk to sixty of water. It had come to the verge of starvation on its diet. In the other case a baby eight weeks old had been ordered by the family physician, in the heat of summer, six ounces of unsterilized milk with Mellin's food every two hours. The child died of cholera infantum. Both these physicians were in active general practice, and I dare say that the children who had fallen victims to their ideas on infant-feeding could be counted in large numbers. And these are but single examples of a large class.

The disadvantages attaching to the use of cow's milk—the only practicable substitute for human milk—in the feeding of infants may be summed up as follows:

Cow's milk differs in the proportions of its chemical constituents from human milk.

Cow's milk, as ordinarily obtained, especially in summer, swarms with many varieties of microbes, and is contaminated, perhaps, with the products of their activity. It is, moreover, acid. Human milk, on the contrary, is drawn from the breast and received into the infant's stomach sterile. It is alkaline.

Cow's milk can be administered in almost unlimited quantities and at any interval. Human milk is supplied by the mother's breast, in quantities and at intervals suited to the demands of her infant's economy.

Each of these factors could defeat an attempt to nourish an infant on cow's milk alone; but, operating together, as has usually been the case, it is little wonder that artificial infant-feeding in the past was one long record of failures. To the theoretical student of the subject at present it might seem that every difficulty has been removed. With the knowledge obtained a comparative chemical study of cow's milk and human milk, notably by Arthur V. Meigs's work, one can construct, with cow's milk as a basis, a correct chemical imitation of human milk.[1] By the labors of

[1] Read before the College of Physicians of Philadelphia, January 7, 1891.

[2] In exact number as follows:

1887—23,140 living births, 5296 deaths under one year.
1888—25,163 " " 4977 " " " "
1889—26,244 " " 5268 " " " "

[1] The future has much to teach us in the comparative chemistry of proteids in milk. In Meigs's formula, that which is designated by the loose and incorrect term "casein," but which should be known as caseinogen, is reduced to the proportion of this substance usually found in human milk. But the information which physiological chemists must, before long, furnish practical workers in

Jeffries, Soxhlet, and others, the chemical imitation of human milk can be administered in a sterile condition. And by the investigations of Snitkin, L. Emmett Holt, and others, we have definite and correct ideas in regard to the quantity of food to be given at each feeding, the interval that should elapse between the feedings, and the rate of increase in quantity required from month to month. With such knowledge, which is but recently available, it would seem possible to construct an artificial infant-food equal in all respects to the mother's milk. But the confidence inspired by this idea would sooner or later be rudely shaken in practice.

For some time my own experience led me to hope that we had at last secured the ideal infant's food. My system, based on Meigs's and Rotch's work, with sterilization and adjustment of quantity and interval to the infant's age, is expressed upon the card which I had printed for distribution among those of my patients who required it.

CARD NO. 1.

1. Have ten bottles prepared clean every morning.
2. Put in each of them, through a clean glass funnel:

Cream	dr. iv.
Milk	dr. ij.
Water	oz. j.
Milk sugar	gr. l.
	[One measure].

3. Stopper the mouth of each bottle with dry baked cotton, and sterilize for twenty minutes.
4. Set aside to cool.
5. Add lime-water, dr. ij, to each bottle before use.
6. Apply a plain rubber nipple to the bottle.
7. Warm to blood-heat in warming-cup.

For a time, I say, this plan yielded the most gratifying results. One child, in particular, I recollect. It weighed ten pounds at birth. The mother had fever for about three weeks after delivery, from an infected endometrium, during which time the child was kept upon the bottle. After a while the mother's milk gradually increased in quantity, so that at the end of the first month the infant was fed at the breast, with the exception of a bottle morning and evening. A week later the child was seized with acute dyspeptic symptoms and diarrhœa. I attributed the trouble to the artificial feeding, which I ordered discontinued; but the child grew worse. The mother's milk was then analyzed, and found to contain but 0.8 per cent. of fat, with 1.17 per cent. of casein. The artificial feeding was resumed, and the little patient at once picked up, gaining from that time an average of two pounds a month up to the last report that I received at the sixth month. But soon there came a succession of failures. The most conspicuous was in the case of a healthy infant born at term of exceptionally healthy and well-developed parents. From the first it was necessary to give this child the bottle. It did not thrive. On the contrary, by the third week it had become obstinately constipated and was visibly wasting away. From my own observation, and from what I had heard of others' experience, I had already learned to suspect that sterilization is the disturbing factor in the effort to produce an ideal infant-food. Consequently, the infant was taken from the mixture described on the card, and put upon condensed milk (1 part), water (12 parts), and cream (1 drachm to an ounce of the mixture). The change in the child's condition was rapid and striking, and at the third month, when I saw it last, it had become in weight, color, and appearance as fine a specimen as one would wish to see.

About this time I assumed charge of the children's wards in the Philadelphia Hospital, with the determination to learn what change sterilization caused in the chemical imitation of human milk and to find a way, if possible, of obviating the alteration. On first thought it seemed that the trouble lay in a disturbance by heat of the chemical constitution of the mixture. It had been asserted that, in boiling, milk loses 3 per cent. of gases (CO_2,N,O) and that 20 per cent. of the albuminoids is coagulated in a thick scum upon the surface (Townsend), and I thought that sterilization might also be productive of a somewhat similar chemical change. But on further consideration, and especially after reading two very interesting articles by Béchamp,[1] it seemed more reasonable to suppose that the unfavorable action of heat upon milk consists in a coagulation of the albumin, especially that around the fat globules, making them, already tougher than in human milk, still more tough and difficult of disintegration in the process of digestion. The question, however, could only be settled by a careful chemical study, and this Dr. Hiestand very kindly undertook for me. The interesting report of the work, just concluded, follows in full:

NOTES ON THE EFFECT OF STERILIZATION UPON MILK.

"To determine what changes occur in milk as a result of sterilization, analyses were made of numerous samples of normal cow's milk and of the feeding mixture used by Dr. Hirst.[2] The results of these experiments showed no change in the total solids of

regard to the relative proportions of lact-albumin in the two milks, and as to the quantity of whey-proteid which appears on the formation of the curd or casein, may, and I think will, necessitate a change in the formula for constructing a chemical imitation of human milk. In this connection see the interesting article by Halliburton, Journal of Physiology, xi. 6, November, 1890.

[1] Bulletin de l'Académie, 3d série, t. xx. p. 718 and Ibid., t. xxiv. p. 214. Béchamp shows that the fat globules in milk are surrounded by a pellicle of albumin; that in human milk this pellicle is more delicate, and more distensible, than in cow's milk.

[2] See card No. 1.

any sample examined before and after sterilization. The amount of albuminoids, fat, sugar, and salts in a given sample of normal milk, or of the feeding mixture referred to, may be recovered after sterilization without loss.

"Subsequent experiments show, however, that the organic constituents do undergo certain important alterations in condition which bear upon the question of assimilation. Observations were directed mainly toward the condition of the albuminoids, fat, and sugar, with the following results:

"I. ALBUMINOIDS.—A. Sterilized and unsterilized portions of a milk-sample were saturated with magnesium sulphate, the precipitated casein separated by filtration, and the albumin estimated in the filtrate. The unsterilized sample yielded 0.33 per cent. of albumin; the sterilized only 0.013 per cent., about 96 per cent. of the albumin, in the latter case, having been coagulated by heat and carried down with the casein.

"B. The percentage of casein in the foregoing sample of milk was 3.27, as shown by a calculation based upon the total nitrogen in the milk. On account of the difficulty of separating the coagulated albumin from the precipitated casein, no observations were made on the condition of the casein before and after sterilization.

"A number of experiments to determine the changes in casein after boiling have been undertaken by various observers; but, thus far, the results have been too conflicting to be reliable. Soldner (quoted in the report of the United States Department of Agriculture, 1890) gives some notes made by Hammersten to the effect that the casein in milk is changed by a temperature of 212° F. The evidence seems to be that the calcium in milk is in some way combined with the casein, and that this combination is modified by a boiling temperature. The exact nature of this change is not well understood. It seems probable, however, that an alkaline calcium compound is formed, which may overcome the natural acidity of the milk. This would explain the altered behavior of rennet in sterilized milk. The experiments incident to his report corroborate those of previous observers and show that sterilized milk coagulates with rennet much less promptly than unsterilized milk. The addition of a small quantity of acid is found to restore to the milk its coagulability by rennet, probably by overcoming the alkalinity produced by boiling.

"II. FAT.—After sterilization the milk was covered with what appeared to be a thin layer of butter-fat. To ascertain whether any fat-globules were freed from their albuminous envelopes, a portion of sterilized milk was rotated in a centrifugal machine for several minutes, along with an equal quantity of unsterilized milk in a separate flask. It was observed that a noticeable proportion of free butter-fat was obtained from the sterilized sample, while the unsterilized sample yielded only the usual layer of cream. This and the following experiments show, however, that only a small proportion of the fat is freed, and that, on the other hand, the globules not liberated have their envelopes so altered by heat as to lessen their tendency to coalesce:

"*a.* Sterilized and unsterilized portions of milk were churned, and a note made of the time required to form appreciable amounts of butter. The unsterilized portion yielded butter in one-third less time than the sterilized portion.

"*b.* Sterilized and unsterilized milk shaken up with equal parts of ether and petroleum spirit, and allowed to stand for twenty-four hours, showed, by microscopical examination, no obvious variation in the size or condition of the fat-globules.

"III. SUGAR.—Examinations of milk before and after sterilization were made both by polarimetric and reduction methods. It was found that the whey of the unsterilized sample had a dextro-rotatory power appreciably greater than that of the sterilized sample. The actual figures for the amount of sugar solution taken were, for the sterilized sample, 3.47, and for the unsterilized sample, 3.87. These figures were confirmed by duplicate analyses. The comparative reducing power of both samples on Fehling's solution was, however, but little affected, the slight differences noted being within the limits of experimental error. The exact nature of the change indicated by the polariscope has not been determined.

"The following is an epitome of the ascertained results of sterilization:

"1. Albumin coagulated.

"2. Casein less readily precipitated by rennet than in normal milk. Acid corrects this condition.

3. "Fat is freed to a slight extent; fat not freed has a lessened tendency to coalesce.

"4. Sugar undergoes some change, as shown by its lessened dextro-rotatory power.

"The considerations suggested by a knowledge of the foregoing facts are:

"I. The coagulation of milk-albumin by sterilization may render the milk more difficult of digestion.

"II. Sterilization interferes with the coagulability of milk by rennet, and presumably, therefore, with its digestibility by the gastric juice. [This scarcely agrees with our present ideas.—B. C. H.]

"III. Free fat, as found in sterilized milk, is probably not readily assimilated in infant-food. The fat not free, being enclosed in a less-easily destructible envelope, is probably slow of digestion.

"For valuable assistance in conducting these experiments I am greatly indebted to Mr. William Beam. My thanks are also due to Dr. Henry

Leffmann for the use of his laboratory, where the experiments were made.

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"Demonstrator of Chemistry in the Woman's Medical College of Pennsylvania"
"PHILADELPHIA, December, 1890"

Being sure that the chemical study would bear out the theory in regard to the effect of heat upon the fat-globules and albumin, I adjusted the clinical work to this idea as soon as the chemical investigation had been fairly started. The only way to obviate the action of heat lay obviously in a partial predigestion of the milk and cream; and the only agent at our command to accomplish this result is pancreatin.

The chemical study takes no account of predigested food, as Dr. Hiestand and myself were working independently at the same time, and were ignorant of each other's work till the whole was completed. One of the methods, described in the report, of comparing sterilized with unsterilized milk suggested to me the following idea: If predigestion with pancreatin really affects the fat-globule and makes it easier to free the fat, this action should be demonstrable by the different effects of churning upon predigested milk and cream, upon sterilized predigested milk and cream, and upon sterilized milk and cream. In the first, three minutes' churning of half a tumbler of the mixture produced enough butter more than to fill a teaspoon. The churn used is a small tin affair sold in housefurnishing shops for making whipped cream, which it takes ordinarily about four minutes to do. In the second, less than four minutes' churning of one and one-half ounces produced a good-sized lump of butter. In the third, it took about twelve minutes to form butter at all, and there was not as much by that time as had been produced by four minutes' churning of the predigested sterilized specimen.

There remains to be supplied one more chemical link in the chain of evidence. The stools of children fed upon sterilized and predigested sterilized milk should show a difference in fat contents. We already know that a diet of boiled milk is accompanied by an excess of unassimilated fat in the stools.[1] I should expect to find less unassimilated fat in the stools of infants fed upon predigested sterilized milk than in those fed upon simple sterilized milk. I hope to have this point investigated in the near future, or to learn the truth about it from the work of others.

Still retaining the chemical proportions suggested by Arthur V. Meigs (with the single exception of sugar), and observing the rules for quantity and interval suggested by Snitkin and Holt, the following card was drawn up:

[1] Vasilieff: Thèse inaugu., St. Petersburg, 1889.

CARD NO. 2.

[Front.]

1. Have ten nursing-bottles prepared clean every morning.
2. Take
   Cream . . . . . 5 ozs.
   Milk . . . . . 2½ ozs.
3. Put in skillet; add pancreatin powder; heat over alcohol flame for six minutes; stir and sip constantly; *do not overheat.*
4. Of this mixture, put in each bottle 6 drs. (to make 2-oz. bottle). Use funnel.
5. Add to each bottle 10 drs. sugar solution.
6. Stopper the mouth of each bottle with dry, baked cotton, and sterilize for twenty minutes.
7. Set aside to cool.
8. Before use, put bottle in warming-cup; apply nipple immediately before giving it to infant.

Make sugar solution by dissolving 1 oz. sugar of milk (1 powder) in a pint of warm water.

[Reverse.]

Age.	Interval.	Number of feedings in 24 hours.	Amount of food at each feeding.	Total amount in 24 hours.
1st week . . .	2 hours.	10	1 oz.	10 ozs.
2d to 4th week .	2 hours.	9	1½ oz.	13½ ozs.
2d to 3d month	3 hours.	6	3 ozs.	18 ozs.
3d to 4th month	3 hours.	6	4 ozs.	24 ozs.
4th to 5th month	3 hours	6	4–4½ ozs.	24–27 ozs.
6th month . .	3 hours.	6	5 ozs.	30 ozs.
8th month . .	3 hours.	6	6 ozs.	36 ozs.
10th month . .	3 hours.	5	8 ozs.	40 ozs.

The pancreatin powder, for the quantity indicated on the card, consists of

℞.—Pancreatin . . . . 2½ grains.
Bicarbonate of sodium . . 5 "

The sodium salt furnishes the alkalinity desired, so that lime-water may be dispensed with.

The objection urged against the partial predigestion of an infant's food, that, by so doing, one deprives the digestive juices of their natural work, and thereby incurs the risk of atrophy of the digestive glands, while at first sight forcible, is in reality weak. If, by this method, the food-mixture is reduced to a condition in which there is about as much for the child's digestive powers to do as though it were fed on human milk there is no danger of abrogating any digestive function.

The sugar of milk is prepared in less amounts than would be called for in an exact chemical imitation of human milk. I am not sure that I will not still further reduce the proportion. Although sugar from cow's and human milk is chemically identical, physiologically it cannot be so, for I have often remarked the diuretic properties of lactose from cow's milk when given to an infant, and I have verified the discovery made by others that the

lactose of cow's milk can produce a very marked glycosuria in bottle-fed babies.

All the infants in the Philadelphia Hospital during September, October, November, and December that were deprived of their natural nutriment, to the number of twenty-one, were fed after this plan. They were weighed daily and their weight entered upon the weight-sheets which I had printed in the hospital. At first a daily record of their stools was kept, but as none of them became constipated this was not continued. In this connection I must express my thanks to the nurses of the hospital for the faithful manner in which they performed an arduous task.

The loss in these four cases was not due to the feeding. In two it was caused by disease, in two others, I think, by the unfavorable hygienic condition in the hospital. The five remaining children died, and naturally lost considerably in the course of their fatal illness. Two of these children were syphilitic, with extensive skin eruptions, and in one of them severe purulent ophthalmia. One died of capillary bronchitis, the other of very severe entero-colitis. This case was especially interesting, for in spite of the care to sterilize its food, the same symptoms were developed with the same fatal result as though the child had received the worst-infected milk procurable. I believe that in

CHILDREN'S WEIGHT-SHEET.

These weights are given by weeks, and in pounds and ounces, thus, 7.2 = 7 lbs. 2 ozs

Name.	Age.	1	2	3	4	5	6	7	8	9	10	11	12	13	14	16
Clarence Wright,	2 weeks.	8.6	8½	7.15	8.2	7.14	7.0	6.11								
John Powers,	2 "	6.9	6.3½	5.15	6.1	6.3	6.9	5.14	5.13	5.13	5.8					
Walter Jones,	3 "	6.15	7.1	6.14	7.2	7.1	7.2	7.0								
Gilbert Burney,	6 "	6.04	5.15	6.4	6.2	5.10	5.8									
Clara Lyle,	7 "	7.0	7.8													
James Ryan,	2 months.	6.12	6.11	6 13	7.0	6.12	6.7½									
Joseph Marianok,	2 "	8.0	7.12	7.13	7.12	7.11	7.10	7.2								
George Carpenter,	3 "	9.8	9.4													
Elizabeth Bartling,	4 "	8.6	8.10	9.0	9.8	9.11	10.2	10.5	10.10	10.15	11.8	12.0	12.5	12.8	12.12	12.14
Joseph Howard,	7 "	12.12														
James Lappan,	7½ "	11 13	11.15	11.8												
Nellie O'Brien,	8 "	10 3	10.1	9.11	9.9	9.14	9.15	9.13	9.8	10.0	10.5	11.2	11.8	11.8	11.12	11.15
John Roth,	8 "	9.9	9 3	8.6	8.8	8.5										
John Hamilton,	8 "	9.9	9.0	10.3	10.5½	10.0	10.1	10.2	10.1	10.4½						
Nellie Johnson,	1 month.	6.13	6.12	7.3	6.15	6.8	6.8	6.13	6.12							
Florence Embers,	3 weeks.	5.2½	5.11	5.8	5.8	5 5	5.5									
Annie Harrison,	2 hours.	5;9														
Annie Whalen,	2 days.	4.13														
Edward Walsh,	8 months.	10.8	10.10													
Nellie Fries,	4 weeks	3.8½	3.7	0.13												
Lenmon Collins,	3 months.	7 4														

It will be seen by the tables that four children were under observation less than a week. They will not be considered. The remaining seventeen infants were of all ages under eight months, some of them very young. Of this number eight gained in weight, nine lost. Of the latter number four lost only a slight amount.

CASE I.—James Ryan, two months old, lost 4½ ounces in four and one-half weeks. In the first three weeks in the hospital he gained 4 ounces, but in the last twelve days before leaving, during which time he had bronchitis, he lost 8½ ounces.

CASE II.—George Carpenter, three months old, lost 4 ounces in one week and a half.

CASE III.—J. Lappan, seven and one-half months old, lost 5 ounces in two weeks.

CASE IV.—Nellie Johnson, one month old, lost 1 ounce in six weeks. This child gained 9 ounces in the first three weeks, but acquired what was at first thought to be pneumonia, but is now believed to be tuberculosis, with consolidation of the upper part of one lung. During the three weeks of her disease she has lost 10 ounces.

children of miserable development, as in this case, the food, though given sterile, decomposes in the intestinal tract, for it is only reasonable to suppose that, when all other vital functions are performed imperfectly, the digestive juices are insufficient not only to digest the food, but even to keep it from rapid putrefaction. The third infant of this series, Gilbert Burney, died of tuberculosis. The whole upper lobe of one lung was consolidated. Of the other two children, one showed a scar of old ulceration in a Peyer's patch, and several fresh ulcers with congestion of the large intestine and disease of a few solitary follicles. This was undoubtedly a case of entero-colitis from food-infection, to be explained, I think, as in one of the syphilitic children. The second infant presented really nothing in the post-mortem examination to account for the death. There was no appreciable entero-colitis, though the mesenteric glands were slightly enlarged. The body was intensely anæmic and very thin. The child appeared simply to waste away.

Of the children who gained in weight, I would

direct attention particularly to one, Elizabeth Bartling, aged four months, who gained 4 pounds and 8 ounces in three months and a half, increasing from 8 pounds 6 ounces, to 12 pounds 14 ounces. It is a significant fact that this infant was not in the foundling ward at all, but in a much larger, better aired room, and in charge of her mother. She was one of twins, one of which was kept on the breast, the other on the bottle. The bottle-fed baby has gained faster than its sister.

I am conscious of much that is imperfect in this clinical study. My material was numerically small. The condition of the infants, many of them picked up on vacant lots, others with parents in the last stages of phthisis, or in the insane department, was in the main very bad, and the environment in the foundling ward of a large hospital is, at the best, unsatisfactory. On one point, however, I am satisfied, namely, that the work of the nurses and resident physicians was earnestly and carefully done. It is on work of this character alone—careful attention to details of feeding and of observation—that a true system of artificial alimentation can be founded. It is a tedious and a troublesome task, but it will amply repay the hardest work—for the discovery of an ideal infant's food that gives as good results as mother's milk will do as much to save life and prevent disease as Jenner's discovery, or that of Koch, even if it proves all that is claimed for it.

In conclusion, a few words as to the practical execution of the directions on card No. 2 in private practice—small matters, apparently, but they may mean the difference between failure and success. First, in regard to the sterilization. This is undoubtedly a burden to the child's attendant, which it should be the physician's care to make as light as possible. The best sterilizer, in my opinion, for convenience and efficiency, is the Arnold's steam cooker. It is easy, however, to devise some arrangement, in a family too poor to buy a sterilizer, that will answer the purpose. As to the bottles, they should be, according to the card, "prepared clean every morning." I have lately seen somewhere the advice to boil the nursing bottles for an hour every day. It reminded me of the excesses committed in the name of antiseptic obstetrics at a time when, in certain hospitals, the child emerged from the vagina under a carbolized spray. It is only necessary to clean the bottles with a strong soda solution and bottle-brush, and then rinse them out well in hot water. They and their contents are made sterile or chemically clean in the sterilizer at the same time. The bottles should be of a form that permits of easy cleansing; as far as possible from the shapes that are at present most in use. Rotch's bottles are the best in this respect upon the market, but their shape is awkward, and their price precludes the possibility of their general use. A drug firm is at present making for me a feeding-bottle which will have a wide mouth, almost no shoulder, no sharp angles, and shall be of moderate price.

Very careful directions must be given in regard to the predigestion of the milk and cream. The time—six minutes—should be indicated by an open watch before the person who prepares the food; it should never be guesswork. The direction should be given never to let the preparation get hotter than one usually drinks the morning coffee. By the end of six minutes the mixture begins to have a distinctly bitter taste. It always takes a minute or two to distribute the milk and cream among the nursing-bottles; this should be done quickly and everything should be in readiness to begin the sterilization at once, for should the mixture in the skillet remain hot for ten minutes in all, it might be so bitter as to be unfit for use. The steam heat, of course, stops the digestion at once.

It is well worth the trouble, in private practice, to conduct properly the whole procedure once before the child's caretakers.

Finally, the nurse or mother must be told to hold the child in the lap while it is fed and to make it consume some time, about ten minutes, in taking the bottle. This is a matter usually understood, but in some cases it needs the physician's express attention. In all cases it is essential to success. I was much impressed with the truth of a remark made by a lady, once a trained nurse, now the wife of a distinguished man and herself a mother, who said on a visit to the foundling ward recently, "It is not so much science that these babies need as a mother's coddling."

ACUTE DELIRIUM—PROBABLY AN INFECTIOUS PROCESS.[1]

BY JAMES HENDRIE LLOYD, M.D.,
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UNDER a multiplicity of terms a form of serious brain-affection, characterized especially by delirium and rapid exhaustion, has been described by various authors. Some of these terms are typhomania, or Bell's disease, delirium grave, acute delirious mania, and *délire aigu*, by French writers. The older writers were apt to speak of the disease as "paraphrosyne," or phrenitis, thus indicating their conception of the morbid process underlying it. Some modern authors, with their passion for clinical differentiation, have probably included kindred cases of a mild type, under such titles as stuporous insanity and confusional insanity. The type of the

[1] Read before the Philadelphia Neurological Society, November, 1890.

disease is well exhibited in the delirium which attends typhoid fever and other infectious diseases, and the morbid process is probably not dissimilar. This disease, or, properly, class of diseases, has been also described under special names, according to its causation, thus, puerperal mania, post-febrile insanity, post-operative insanity, septicæmia, exhaustion from overwork, sewer-gas and other poisoning, have all included this acute delirious condition. A few physicians fall back upon the word "meningitis," to explain the disease, and it may be a question whether a few of the cases have not been called delirium tremens.

Some authors include in delirium grave only rapidly-fatal cases, while others include less severe, long-continued, and curable cases. The mere clinical distinctions are too often arbitrary, and are entirely uninstructive as to the exact causation. I have seen one case which proved fatal in less than three days, while another extended over several weeks, and recovered. In both of these cases the condition was a typical delirium, with dried secretions, brown tongue, rapid pulse, variable temperature, and exhaustion, with emaciation. There is no sufficient reason to place these cases in separate classes, with distinctive names, simply because one died and the other lived. So, again, the type of delirium may vary, as may also its severity. One case may be of a low, muttering form, tending to stupor, dementia, and death, as has been reported after some of the exanthematous diseases, such as scarlatina and smallpox; another may exhibit rather a confusion of intellect with active hallucinations, and persist for weeks, with final recovery, as is frequently seen after childbirth. This fact does not seem to me a sufficient reason for a classification which shall be final, especially as the active causation in each case is practically as yet unknown.

The symptomatology of these cases varies, but presents a sufficient number of resemblances to constitute a simple broad class. The mental symptom is usually wandering delirium. In milder cases the attention can be momentarily arrested, and a question or two answered; but the mind soon lapses into more or less complete object-unconsciousness. Subject-consciousness is much confused, and is apt to be dominated by visual and aural hallucinations. From this stage all grades of delirium to complete stupor and coma can be observed. The physical state accompanying the delirium is apt to be that which is spoken of generally as "typhoid," and grows more marked the longer the acute stage lasts. The tongue becomes dry and brown, and sordes form. The breath is offensive. The pulse is rapid and compressible. There are muscular unsteadiness, tremor, and incoördination. The temperature occasionally rises, but not in all cases, and in my experience pursues no regular course. The gastro-intestinal tract in my cases was not involved, except that there were aversion to food and retention of fæces. In puerperal cases of this type there may be foul lochia and pain on pressure over the uterus, but these symptoms are not present in every case. In two well-marked cases of delirium following childbirth I could discover no focus of septic infection, although I searched for it. Still, I believe they were the results of infection. In one puerperal case seen in the Pennsylvania Hospital for the Insane, under Dr. Chapin's care, the patient's active delirium came on within a month after confinement, and was marked by anæmia, emaciation, and a brown dry tongue. She had had at first fleeting, delusive ideas, and rapidly became immodest, restless, and troublesome in every way. She talked incoherently, but later had a fixed, staring gaze, with dilated pupils, and would not respond to questions. There was no abdominal soreness and no fever. The blood-count showed that the corpuscles were reduced twenty per cent. It is usual to call such cases "puerperal insanity," yet their likeness to acute delirium, especially when they are severe, is too great to be ignored. Such cases sometimes run a very rapid "typhoid" course, and die, but in the main the prognosis in puerperal cases, if taken in time, is good.

In some of the cases, called post-febrile, such as are seen after scarlatina, smallpox, and typhoid fever, the early stage of the acute delirium is coincident with the acme of the infectious process. The mental condition at first is looked upon as merely the delirious reaction so commonly seen in the exanthemata, and is no doubt identical with that reaction; but as the specific disease subsides it is found that the brain-cells have not recovered from the toxæmia, and a psychosis results which may pursue a variable course, either as confusional insanity, insanity of fixed delusions, or, rarely, dementia. These cases are of special interest, because no one doubts that they are due to infection; hence, in them the action on the convolutions of specific infectious products, probably ptomaïnes, may be observed, and important hints and analogies supplied for the understanding of similar cases in which infection can only be surmised, and not demonstrated.

In the fatal case which Dr. Tull has reported (see page 127), and which I saw with him in consultation, the onset was very sudden and the course very rapid. In this case there was no known source of infection. We could exclude with reason alcohol, syphilis, uræmia, lead, and probably all the known zymotic poisons:

The patient had worked in a district which possibly was malarious. He was seized on a Saturday,

at midnight, and died on the following Tuesday morning, living less than sixty hours after the onset. He was an unusually robust man, less than forty years old, one whom it was not easy to kill. He was at first acutely maniacal, with what seemed slight hysteroidal symptoms, and his attention was held with difficulty and but for a moment. There was great motor disorder, so that he had literally to be held in bed. He struggled, resisted, spoke not a word, had stareing eyes, refused food, and seemed at times rather emotional and histrionic. His pulse at first was strong and good. His temperature varied, without regularity, and once rose to 103°. It was sometimes normal. His urine was not albuminous. He sank rapidly, as if under the influence of some baleful intoxicant, became comatose, and died. The autopsy revealed to the naked eye nothing but white streaks in the perivascular sheaths, which probably indicated the first attempts of nature to meet an invading irritant poison.

The diagnosis of some of these cases is extremely perplexing:

In a case which I saw with Dr. Gittings, in West Philadelphia, the patient, a man aged about forty-three years, lay for more than two weeks in a stuporous delirium. He was aroused with difficulty. His tongue was dry, brown, and hard, and his pulse feeble. There were absolutely no other symptoms to guide in forming a diagnosis. His temperature seldom, if ever, rose above normal. His urine was frequently examined, both chemically and under the microscope, with negative results. Alcohol and lead were not factors in the case. Syphilis was excluded as well as it usually can be, and the post-mortem lesions did not indicate this poison. The general appearance of the patient was somewhat like that of a man profoundly ill with typhoid fever, but the afebrile temperature, the absence of abdominal symptoms and eruption, negatived such a diagnosis. There was no history or appearance of trauma. An examination of the eye-grounds by Dr. de Schweinitz gave no results. At the autopsy the brain was removed before the great vessels in the thorax were cut—an important precaution. There was undoubtedly a great deal of blood in the brain, but a careful inspection of the vessels, membranes, and convolutions revealed nothing which could in any way explain such a grave disease. The longitudinal sinus contained a long, slender thrombus, which, however, did not occlude the calibre of that canal.

In a case under my care, in the Philadelphia Hospital, a protracted delirious state came on in a patient who had been for a year in a simple maniacal exaltation. I do not, of course, call it a case of acute delirium in the sense in which that term is usually employed. It was rather a delirium engrafted upon a mania. The case had all the usual accompaniments: altered secretions, rapid pulse, muttering incoherence, etc. The interesting fact is that *after* this delirium had been established for some time the patient had a diffuse cellulitis of the leg, erysipelatous in appearance. There was evidence here of some infection, but of what character I could not determine.

The symptoms of meningitis, or meningeal irritation, are seen in some of these acutely delirious cases. The following interesting case was admitted into the Pennsylvania Hospital for the Insane, where I examined the patient and signed the certificate. I am indebted to Dr. Chapin for notes of the case:

The patient was a woman, aged twenty-one years, who was reported to have had a "malarial attack" two months before admission. This illness left her in an impaired mental condition, marked by agitation, mental confusion, insomnia, and emotional disturbance. She rapidly grew worse, and was admitted to the hospital with a diagnosis of acute delirious mania. When I saw her she was in a typical delirious state. She was entirely incoherent and inattentive, muttering, and expressing what seemed to be fragmentary, delusive ideas. She evidently had hallucinations. The prevailing tone was apprehension and distress. She was restless, and was kept in bed with difficulty. Her tongue was coated, bowels constipated, pulse 105, and temperature 99⅘°. The pupils were dilated. A very marked symptom was the muscular spasticity, with convulsive movements in the hands and arms, and much exaggerated patellar reflexes. Her urine was examined in the hospital, and showed neither albumin nor casts. During the last week of life her temperature rose steadily day by day, until just before her death, when it was 104⅘°. Her skin was hot and oily. The loss of object-consciousness in this case was almost complete. An autopsy was refused.

It is notable in this case that hyperpyrexia was present, as well as a general spastic state of the muscles approaching a convulsive condition. The exact nature of the primary infection was apparently not made out It was called "malaria," but this term is so loosely used in the profession that often little meaning can be attached to it.

In the brief review here presented of these cases it has not been my aim to form any theory of causation. No one theory can cover all these cases; no one special *materies morbi* can explain cases so widely separated as a delirium happening after smallpox on the one hand, and a puerperal delirium on the other. Many of the causes put down in the text-books are entirely inadequate, in my opinion, to account for the delirium in the vast majority of cases. These causes are anæmia, exhaustion, mental and moral shock or strain, such as grief, fright, worry, etc. These may be contributory causes, and in the case of blood-impoverishment we have, probably, an important predisposing cause: but it seems to me that we want something more definite and tangible than worry to explain the death of a strong man in less than three days from the onset of the disease; something more

reasonable than an uncertain "malaria" to explain an acute delirious mania, with symptoms of meningitis, in a vigorous young woman.

In the cases following the exanthemata, and in the puerperal cases, an infectious poison is probably the immediate exciting cause. Clark gives details of forty patients with puerperal insanity, of whom nearly 50 per cent. had septicæmia or simple inflammatory trouble about the uterus. Clouston's puerperal cases showed a much higher range of temperature than any other class of cases—an "alarming result," as he thinks, of septic and inflammatory disorders. Since the time of Hippocrates this septic delirium following childbirth has been noted. The father of medicine himself reports a fatal case following a delivery of twins, in which case there were suppressed lochia, chills, insomnia, incoherence, convulsions, suppression of urine, coma, and death. Guislain distinguishes what he calls puerperal meningitis, which is characterized by sweats, fever, abdominal pain, and coma, and is evidently a septic delirium. With an increasing knowledge of septic disorders in childbed we shall probably hear more in time to come of sepsis as a cause of insanity in the puerperium.

But the cases in which we have a sudden explosion of delirium, without any recognizable source of infection, are not so easily explained—in fact, are not yet explainable at all. We are left here to speculation.

In Dr. Tull's case we found a perivascular engorgement. Possibly, ere long the case would have presented distinct pus-formation, and pus in the brain means infection. We stand to-day probably upon the border-land of a new domain in pathology, physiology, and, above all, animal chemistry.

It is premature to dogmatize, but it has appeared to me from a clinical study and comparison of this group of cases, that the symptoms point in the direction of an irritant poison. In his recent work Bevan Lewis has given the most complete and profound study of the morbid anatomy of the brain which has appeared in our language. It is noteworthy that he gives special prominence to the lymph connective tissue, and its "scavenger cells," which are found gorged with products of "intercellular digestion" especially in cases of rapid waste and deterioration of the cortical and neural tissues.

The microscope, however, will, probably, not continue to be the court of last resort in the study of mental disease. It is, after all, only an aid to the eye, and does not show things essentially invisible to the eye. It, probably, cannot show the vito-chemical reactions of the nerve-cell better than it shows the reaction between an acid and a base. It has already demonstrated so often, and in the hands of so many, the slight morbid changes in capillaries and neuroglia, as seen in these early fatal cases, that to repeat them again and again does not add to knowledge.

In the language of Montaigne: "*Je raconte, je ne juge pas*"—I relate these cases, I do not give final judgment upon them.

REMARKS ON ACUTE DELIRIUM.[1]

BY JOHN B. CHAPIN, M.D.,
SUPERINTENDENT OF THE PENNSYLVANIA HOSPITAL FOR THE INSANE, PHILADELPHIA.

HAVING been kindly asked by Dr. Lloyd to make some remarks upon the cases reported by him, it has seemed the better course to offer some general observations which they suggest. Few physicians from their own experience can have much to say of *acute delirium* (a term which Dr. Lloyd and Dr. Tull seem to prefer). As the disease is of rare occurrence, few physicians see a single case in the whole course of their practice, and typical cases are of infrequent occurrence in hospitals. Dr. Lloyd, like others, may have been surprised at the sudden onset of grave symptoms of an anomalous and unaccountable character, and at the steady progress of the disease to a speedy and fatal termination. I confess to having experienced such a feeling, as well as chagrin that all available measures applied for the relief of the patient seemed of no avail to prevent a fatal result.

As early as 1849, the French (Brierre) applied to cases of this class of diseases the term *acute delirium*. Dr. Luther Bell, during the same year, reported a number of cases with symptoms which he declared made out "a new form of disease," or a disease not previously recognized. From the rapid "emaciation," the appearance of sordes on the teeth and mouth, and the low condition of the patient, the term *typho-mania* was also used, suggested probably by the prevalence of typhus in the larger seaport towns at that time, and a similarity to the asthenic character of that disease. The Germans (Dr. Schüle, 1868) have recognized the group of symptoms presented by Dr. Lloyd's cases as a distinct form of disease to which they have applied the name *acute delirium* while in England, Dr. Blandford and others have used the term *acute delirious mania*, which seems to me appropriate and consistent with our usual nomenclature. The disease is a mania accompanied with active delirium.

If Dr. Lloyd has used the term *acute delirium* from what was apparently in his mind as to the possible etiology of these cases, a new field of broader speculation and investigation certainly is opened. That cerebral disease, with psycho-motor disturbance, has appeared even in an epidemic form from the

[1] Read before the Philadelphia Neurological Society, November 24, 1890.

inhalation of sewer-gas and the effluvia of old privy vaults is well known. The so-called meningitis at the Pittsfield (Massachusetts) Seminary, and at Aurora, New York, was due to the ventilation of privy vaults into the school apartments by closed passages. I myself have observed 40 out of 300 persons in one building prostrated in the course of twenty-four hours with cerebral and spinal pains, vomiting, sense of weakness, and several of the cases with delirium. So sudden was the onset that the illness was suspected at first to be due to poisoned food, yet was traced to the introduction of sewer-gas through an unused trap.

The frequent occurrence of cerebro-spinal and spinal meningitis in Philadelphia is of comparatively recent date, and is, I believe, to be largely attributed to the existence of drainage wells in houses and defective ventilation of sewer-pipes and water-closets.

While there is a strong presumption that meningeal disturbance, attended with delirium and motor excitement, results from infectious causes, *acute delirium* or *acute delirious mania* cannot be said to be caused by meningeal inflammation. The post-mortem appearances are of such a negative character that the centres of life, intellect, and sensation would seem to be mainly involved, no traces of physical disease and change being observed, as a rule. Yet, in the present state of our knowledge, with clues leading to new lines of scientific investigation, can any one wholly ignore the suggestion that Dr. Lloyd has presented?

While the sudden appearance of the prominent manifestations may suggest the existence of an active infectious process, it must be borne in mind that when we obtain a history it will be ascertained there has been a strain from overwork, worry of some kind, prolonged insomnia, or some moral shock. In other cases there are excesses and possibly other causes of which we can ascertain nothing. I can say of one case that a winter of social dissipation in Washington was a part of the history, and of another, frequent childbearing and constant domestic cross-purposes resulting in exhaustion and insomnia. The death of Horace Greeley from *acute delirious mania*, after the mental strain attending his political canvas is well remembered. The cases that I recall occurred in persons apparently well-nourished physically. It could not be said of them they were constitutionally neurotic, yet nervous heredity has often been noticed. After a few days of noticeable hurry and worry about small, non-essential things, which may not be recalled until the disease has developed, and may not even attract attention at the moment, and without pronounced prodromic symptoms, the patient may awaken in a confused or delirious state. There has been no chill, vomiting, headache, or spinal pains. The attention of the patient may be roused and held for a brief period in exceptional cases, but as a rule even this is impossible. The mental state is one of profound confusion, or absorption in centric ideas. Hallucinatory disturbance of several, or all, of the senses is the prominent manifestation. There is also excessive motor disturbance—the patient is in constant motion—and there is frequently a tendency to gyratory motions. The delusions are those of fear, apprehension, or suspicion, of persons and food, and there is much difficulty in the administration of nourishment. The patient does not usually talk in a loud and demonstrative manner, but in a quiet tone, with little reference to surrounding objects or persons—much like the conversation at one end of a telephone. The total absorption of the mind of the patient in centric ideas, indifference to all surrounding objects, and inability to attract the attention in these and all similar cases, are unfavorable prognostic signs; but when the delusions have reference to and are connected with surrounding objects and persons, the case is more hopeful. The pulse shows a wide range, as from 100 to 120 and 140 in a brief interval of time. The temperature reaches 104°, or even 106°, usually 100° to 102°.

There are also distinct remissions, like lucid intervals of a few hours' duration, in which the patient seems for the time rational, and the pulse and other conditions resume a normal state. Possibly, but certainly rarely, the disease may suddenly end in this manner. If, however, there are exacerbations and remissions of all of the symptoms, if there are profound delirium and insomnia, if the attention cannot be roused, if the several senses are more or less involved, if there is great motor disturbance, if, at short intervals, there is a great variation of the pulse, if the fingers tend to the palms, the prognosis is decidedly unfavorable. Death may take place in two or three days; but if life can be preserved for from ten days to two weeks, the stage of acute delirium subsides, but the maniacal stage continues, with chances that in one or two months recovery will take place. It is the general experience that when death does not occur in the early stage recovery follows rapidly and completely, and without a stage of succeeding dementia.

The probabilities in a case of acute delirium, or acute delirious mania, are against recovery, and this fact should be borne in mind if the question comes up whether the patient should at once be sent to a hospital, or whether there should be a delay of a few days. Undoubtedly the mortality from this disease was formerly greater than now. The rapid waste of tissues, the dry tongue, the sordes on the teeth, and the general asthenic condition, are more promptly met now by a liberal administration of food and stimulants. Then, again, the new hypnotics can

be used safely and effectively, without the necessity of administering drugs, that sometimes do more harm than good by intensifying the delirious condition, and developing a new pathological state. I have seen hyoscine produce sleep, and quiet the excessive restlessness and struggling characteristic of this disease.

In the progress of the disease there will be observed a tendency to sluggish circulation—a stasis of the capillary circulation. This is a symptom of serious import, indicating the extent to which the vasomotor system is involved. It is important to be able to combat a tendency which is a precursor of a fatal termination. I have in three cases seen this condition removed by packing the patient in hot wet sheets. In each case the supposed hyperæmia of the brain seemed to be relieved, the general circulation was improved, there was diaphoresis and a free discharge of urine.

In our present state of knowledge, anything that may be said of the pathology of a disease that leaves no appreciable traces is partly speculation. The cases of this disease furnish a history of strain from overwork, worry and anxiety, shock, or of some profound emotional disturbance at some period, such as may produce an arrest, alteration, or disorder of nervous nutrition. As the deficient nutrition of the nervous mass and the asthenia and collapse which characterize these cases are due to a deterioration of the quality of the blood, and to the state of the cerebral circulation, the state of the patient, as we observe him, is that of mental and physical incoördination. There are successive explosions of psychic and physical force, such as may be supposed to arise from the suspension and disorder of the inhibitory functions. As the tendency is to death from asthenia, the indication for treatment from this point of view is to support the patient during the existing paroxysm, and the prolonged use of tonics and rest during and after convalescence.

A CASE OF ACUTE DELIRIUM.

BY M. G. TULL, M.D.,
OF PHILADELPHIA.

THE patient, Charles P., aged thirty-nine years, was a farmer until eight years ago; since then has managed a switch-board in the tower of the Philadelphia, Wilmington and Baltimore Railroad, at Gray's Ferry Station, and at the same time carried on a grain and grocery business. When nine years old, he fell from an ox-cart, which caused bleeding from the left ear for one day, and the ear remained permanently deaf. His subsequent history was that of unvarying health. In appearance he was a man of typically fine physique.

On August 16th he spent the day at Atlantic City, where he stayed in the water for four hours.

On August 24th, at 6 A. M., I was called to see him. I found him tossing to and fro on the bed, flinging his arms about, and with a vacant expression of the eyes. His only symptom of distress was a constant tendency to move his hands toward the bladder. He was apparently semi-conscious. Thinking that the condition might result from retention of urine I catheterized him, and found the urine normal in color, odor, and quantity. I carefully examined for albumin, with negative results. In fact, I could find no evidences of organic disease, and was at a loss for a diagnosis. This condition continuing during the morning, I suggested a consultation with Dr. J. Hendrie Lloyd in the afternoon. Dr. Lloyd pronounced the symptoms hysteroidal mania, and he also could find no evidence of organic disease. The pulse during this time varied from 75 to 88, the temperature from 100° to 101°. His skin was very dry. I had given $\frac{1}{4}$ grain of morphine with $\frac{1}{60}$ grain of atropine hypodermically to control restlessness. I then employed steam-baths, and pilocarpine both hypodermically and by the mouth. After the steam-bath was continued for two and a half hours his skin became moist. The condition of the patient continuing unrelieved, in deference to the apparent wishes of the family, who were convinced that there was some ear-trouble, because previously to the attack he had complained of pain about the right side of the head, I requested Dr. J. B. Deaver to see the case. He joined Dr. Lloyd and myself in consultation, Sunday night, August 24th. We agreed that the symptoms were simply maniacal, and that there were no evidences of organic disease.

On Monday, August 25th, the same conditions persisted, except that the patient seemed more conscious. Between the paroxysms he was able to sit on the bed and urinate, but he passed very little urine and with great difficulty. His temperature varied from 100° to 103°, and pulse from 75 to 88, his respiration averaging 24. During this whole period the conjunctival reflexes were present, and his attention could be partly fixed by a quiet, determined effort. On Monday, at 11 A.M., Dr. Lloyd again saw him with me, and considered that the diagnosis of non-organic disease was confirmed. We again put him on opium and small doses of belladonna by the rectum. During Monday night, August 25th, the patient rapidly became worse. I was sent for at 6 o'clock A.M., when I found him dying, his pulse having increased to 105, and his temperature to 103$\frac{3}{5}$°. At 8 A.M., August 26th, he died.

That afternoon the autopsy, kindly conducted by Dr. J. A. Davis, assisted by Dr. Brinkman, in the presence of Dr. Lloyd and myself, revealed a condition apparently of incipient meningitis. The temporal bone was not diseased.

After the man's death I was informed by his neighbors that his temper had seemed decidedly more irritable and aggressive than usual for some time past, and that he had been working eighteen hours daily.

ON THE RELATION OF ECZEMA TO DISTURBANCES OF THE NERVOUS SYSTEM.[1]

BY L. DUNCAN BULKLEY, A.M., M.D.,
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No careful medical observer or thoughtful student of medicine can rightly doubt or ignore the part which the nervous system plays in relation to many diseases of the skin, although the exact connection and causal relations between nerve-disease and skin lesions may not always be apparent or satisfactorily explained. The intimate association of certain skin affections with, and their dependence upon nervous elements have passed beyond the basis of personal judgment and experience, and rest upon sound anatomical, pathological, and clinical grounds, which are being confirmed and strengthened daily.

Time and space fail us to detail here the proofs of this statement, most of which are more or less familiar to all; the careful and thorough writings of the many observers to be mentioned later on are full of demonstrations of the neuro-pathology of various diseases of the skin, and the present writer has on several occasions elaborated the subject, giving clinical illustrations of the same.[2]

It suffices, therefore, for our present purpose to accept the fact that the nutrition of the skin and of the cells composing its structure is under direct nervous control, so that under proper nerve-influence the processes of health are carried on, while under perverted nerve-action many pathological changes may take place in the skin, as well as in other tissues.

Fully accepting this basis of a possible neuro-pathology of lesions in the skin, it remains for us to study how far clinical and pathological facts point toward this element of causation for the eruption, phases of eruption, or disease to which the name of eczema is given.

In approaching the subject of the relation of eczema to disturbances of the nervous system, the questions naturally arise: What is eczema? What is it that constitutes the disease under consideration? What is it that determines that the skin lesions seen in any particular individual are eczema, and not some other affection? What are the real essence and nature of the disease? Is it purely a skin affection, consisting of certain changes in the cutaneous tissue, from many causes, or is there some underlying condition, habit, or state of the system, which determines the pretty uniform set of cutaneous symptoms which we call eczema? It would seem that these questions should be definitely answered before we could make any great progress in a study of the disease or its treatment, and it is hoped that the present discussion may aid somewhat in their solution. But everyone familiar with dermatological literature knows that they have been the theme of countless discussions, and that widely-divergent opinions have been expressed by those of large experience in the disease in all countries, so that even to-day there is no universally accepted view as to the ultimate nature of eczema.

It would be out of place to enter here at all fully into the discussion of these topics, or to consider at length the morbid conditions of the skin to be included as eczema, but a few words must be allowed in order to understand rightly the basis of our study.

We know and recognize eczema by its cutaneous phenomena, for it cannot be said that it has any other absolute symptoms than those found upon the skin. But still, polymorphous and *bizarre* as those appearances upon the skin often are, simulating at times many other quite different cutaneous affections, there is a group of symptoms which are universally recognized as belonging to eczema, and of a thousand cases so named by a competent dermatologist, the diagnosis of very few would be questioned by others similarly qualified. A brief definition which would cover every case of eczema, or a short description of its forms and phases as seen in persons of various ages and on different parts of the body, would be very difficult to give; and, after all, it is only the composite picture, including many features belonging equally to other affections, that we designate as eczema.

But, in connection with various cases of eczema, we often meet with other phenomena also than those upon the skin—that is, we find disturbances or disease of other organs of the body, which we learn by clinical observation to associate more or less closely with the disease under consideration, and which are found by experience to be of importance etiologically. These, furthermore, are seen to improve or disappear with the hygienic and general treatment or measures directed to the eruption, and yet none of them can be called absolute symptoms of the disease, or essential features of the eczema. The elements referred to are such as anæmia, lithæmia, the strumous or gouty habits or conditions, high arterial tension, and also various derangements of

[1] Prepared by special request, to open a discussion on eczema at the Tenth International Medical Congress, Berlin, 1890.

[2] The Relations of the Nervous System to Diseases of the Skin, Archives of Electrology and Neurology, New York, November, 1874, and May, 1875.

Two Cases of Exophthalmic Goitre and Chronic Urticaria, etc., Chicago Journal of Nervous and Mental Diseases, October, 1875.

A Clinical Study on Herpes Zoster, American Journal of the Medical Sciences, July, 1876.

Herpes Gestationis, etc., American Journal of Obstetrics, February, 1874.

Clinical Notes on Pruritus, Journal of Cutaneous and Genito-Urinary Diseases, 1887.

Alopecia Areata, Medical Record, 1889.

other organs, mainly the digestive tract, liver, and kidneys, together with certain *nervous phenomena;* all these may be well- or ill-defined, and may either obtrude themselves or be discovered only after close investigation.

As remarked before, none of these elements have been proven to belong properly and absolutely to eczema, but still from accumulated clinical experience they must, singly or combined, be considered factors in the production of the disease. If, however, we are pressed to a single answer as to the real nature of the disease, we are obliged to state that we as yet know very little in regard to the true and fundamental cause of eczema: why one person should have it when another individual escapes, who is apparently in the same or worse condition in respect to the derangements of health mentioned, cannot be explained.

The real reason, however, possibly lies in the local condition or constitution of the skin itself, which renders it liable to take on this peculiar form of inflammatory action, either upon the application of external irritants, or from neurotic or other derangement of its nutrition from within. To this condition or state of the individual in whom these changes are likely to take place, under the same or other causes, we apply the term *eczematous*, without indicating thereby any exact or particular disease or known alteration of the system, simply for the reason that we do not know what that is.

I think, therefore, that in studying the relation of eczema to disturbances of the nervous system, or its relation to any other derangements, functional or organic, of any portion of the economy, or in considering the effects of local agents in producing the eruption, we should be very careful to discriminate between predisposing and exciting causes, and give due weight to each: *post hoc ergo propter hoc* is a very seductive and dangerous form of reasoning in medicine, but one which is often unconsciously, or sometimes ignorantly, applied.

Thus, when we see an eruption, which we call eczema, apparently follow or depend upon some nervous disturbance, we should carefully consider (1) if, possibly, it may be only a coincidence; or (2) that the two may depend upon one common cause; or (3) that the nerve-disturbance may have excited the eruption in one previously predisposed thereto; or (4) that the effect of the former may be very remote, putting in action a train of systemic changes which may end in inducing the eruption. In none of these instances could we properly speak of the nerve disorder as the cause of the disease. Even when the eruption seems to follow pretty directly upon some severe nerve strain, shock, injury, or disease, much caution should be exercised in ascribing an immediate causative connection, for how rarely do we find these conditions followed by the development of eczema; and, on the other hand, how very few of the multitudes who suffer from eczema ever exhibit any causative elements pointing toward the nervous system. To my mind the proof is infinitely stronger in regard to many other of the internal and general causative elements mentioned, and in a far larger proportion of cases, than is that regarding the connection of eczema with disorders of the nervous system.

But still, while it is thus urged that caution is necessary before accepting any etiological relationship between eczema and nervous disturbances, it must yet be granted that the weight of evidence is very strong in favor of such relationship to a greater or less extent in certain cases, and that possibly, as our thought is turned in this direction, it may become even more evident than it is at present; and certainly it is wise to regard the neurotic aspect of many cases of eczema in connection with the therapeutics of individual patients.

There is one clinical feature or symptom in eczema which would seem to point to a neurotic element in the disease, even to the most superficial observer, namely, the itching, without which the disease may hardly be said to exist. Not only is the sensation of pruritus a common and severe symptom when the eruption is pronounced, but even before any amouut of lesions are appreciable on the skin, and often long after they have ceased to be prominent, the itching may prove most distressing; and all recognize that scratching to relieve itching is a most prolific cause of the development and increase of the eruption. How far the itching is a primary feature, and how far it results from pressure upon nerve filaments by effused fluid or swollen cells, it is impossible to state; it is certain, however, clinically, that in an acutely-developing vesicular eczema there is much relief from the itching when the fluid reaches the surface, either spontaneously, or by artificial aid; on the other hand, some of the most distressing and inveterate pruritus is found in certain cases of erythematous eczema about the face, where there is but little infiltration of tissue. This latter form is very apt to occur in elderly persons, and in those nervously broken down.

For many years writers have recognized, to a greater or less extent, the dependence of certain cases of eczema upon nerve-disturbance or disease, and in addition to the generalized statements found in text-books to this effect, there are a considerable number of recorded observations in different countries which tend to show the clinical and pathological soundness of this view. It will be our endeavor to classify these observations, and to present, as briefly as possible, such evidence as seems of special interest.

There are so many points to be considered in

studying the influence of the nervous system in the production of eczema, that it is by no means easy to weigh the relative effect of the various nerve lesions or disorders which have been reported in this connection, nor to distinguish clearly the real mode in which the deranged neurotic element operates; at present much relating to the subject is mere conjecture, waiting for verification or refutation with the light of advancing science. But as all work can be best carried on when there is a skeleton basis on which to arrange proof, we may attempt to construct a scheme about which clinical and pathological observation and research may build as opportunity offers.

With this view, and with the material at hand, we will consider the relations of eczema to disturbances of the nervous system under the following heads:

1. Anatomical, physiological, and pathological grounds for such a relationship; and

2. Clinical grounds, based on recorded observations.

1. *The anatomical and physiological grounds* for a nerve-relationship in eczema need not detain us long. All are familiar with the very abundant nerve-supply of the skin, and with its distribution even among and into the cells of the rete Malpighii, and into the cells composing the glands of the skin, and that nerve-fibres are very freely supplied even to the hair-follicles and lymph-spaces, as shown especially by Arnstein.[1]

The influence of the vasomotor nerves, both in the normal regulation of the blood-supply and nutrition, and also in the production of morbid states, is now so well recognized both in regard to the skin and other organs that it needs but to be mentioned.

We are also well aware of the control which the sympathetic system exercises over various processes of the body, healthy and morbid, including the skin and its glands, as has been so carefully elaborated by Eulenberg and Guttman,[2] E. Long Fox,[3] and others. The neurotic aspects of many skin affections have also been admirably worked up of late years by Rendu,[4] Testut,[5] Leloir,[6] Schwimmer,[7] and others,[8] so that now there is no reason to doubt that from anatomical and physiological grounds changes may take place in the skin through the medium of the nervous system.

The direct anatomico-pathological grounds for recognizing a nerve origin of eczema have unfortunately been studied but little, and the subject offers a promising field for future research. The condition of the nerves in the affected skin in acute and chronic eczema, also the state of the conducting nerves and of the cord, likewise that of the sympathetic nerves and ganglia, have thus far been hardly at all studied. The much-quoted observations of Colomiatti[1] are almost our sole basis of knowledge regarding the condition of the cutaneous nerve filaments in eczema. It will be remembered that in a number of cases of eczema, mainly of the papulo-squamous type, he reports finding material changes in the cutaneous nerves, while in cases which had been partially or wholly cured, he claimed that the nerve-tubes had been to a large part restored to health; but he was unable to observe the various changes of regeneration. Leloir[2] states that in two cases of eczema in which he examined the nerves, he found both the nerve-filaments and their sheaths normal; but in a later communication[3] he says that he has verified the observations of Colomiatti.

2. *The clinical grounds* for the assumption of a relationship between eczema and disturbances of the nervous system are much more full and interesting, and have been dwelt upon by many observers.

We may divide our study of this portion of our subject into five general heads, considering eczema as it is related to

1. Neurasthenia or nerve-exhaustion.
2. Nervous and mental shock.
3. Reflex phenomena: (*a*) of internal origin; (*b*) peripheral.
4. Neuroses: (*a*) structural; (*b*) functional.
5. Encephalopathy and myelopathy.

1. NEURASTHENIA OR NERVE-EXHAUSTION.

Neurasthenia, or general nervous exhaustion or depression, must be placed first in the list of neurotic conditions associated with or causative of eczema, since in this is found a predisposing element in very many cases of the disease; moreover, it undoubtedly

[1] Arnstein: Sitz. der k. Akad. der Wissensch., Bd. lxxiv., Oct. 1876.

[2] Eulenberg and Guttman: Die pathol. des Sympatheticus. Berlin, 1873.

[3] Fox: The Influence of the Sympathetic on Disease. London, 1885.

[4] Rendu, Annales de Dermat. et de Syph., vol. v., 1873, p. 412.

[5] Testut: De la Symétrie dans les Affect. de la Peau. Paris, 1877.

[6] Leloir: Recherches clin. et anat.-path. sur les Aff. cutan., etc. Paris, 1882.

[7] Schwimmer: Die Neuropathischen Dermatosen. Wien, 1883.

[8] Thèses:

Triesch: Ueber den Einfluss der Nervensyst. auf Erkr. der Haut. Marburg, 1872.

Hageman: Ueber den Einfluss der vasom. Nerven auf die Hautk. Halle, 1874.

Koster: Die Hautk. und ihre Beziehungen zum Nervensys. Halle, 1876.

Witzel: Ueber den nervösen Char. des Ekzems, etc. Berlin, 1879.

Czempin: Die Theorien über den Einfl. des Nervens. auf d. Ent. der Hautk. Berlin, 1884.

[1] Colomiatti: Gior. Ital. delle Mal. Ven. e della Pelle, 1879.

[2] Loc. cit., p. 164.

[3] Annales de Dermat. et de Syph., 1890, p. 475.

lies at the bottom of many instances in which other nervous conditions act as the exciting cause of the eruption, as will be mentioned later on.

While this term neurasthenia is somewhat indefinite, and as a disorder it may be said to have no pathology, yet practically the condition does exist, especially in America; furthermore, a general nerve-exhaustion, from many causes and taking many shapes, is not infrequently found in a certain class of eczema patients, and does, to all appearances at least, have much to do with the genesis and continuance, or recurrence, of the eruption in many cases.

Clinical proof of this could be furnished in abundance, and numberless cases could be detailed in which eczema, more or less severe and general in character, followed directly upon prolonged nervous strain, such as long attendance upon the sick, distressing or exhausting work of any kind, sleepless nights from worry or anxiety, etc. A few illustrative cases, some of which have already been mentioned in print, by the present writer, may be briefly alluded to:

An unmarried lady, aged fifty-three years, was the executive officer of a charitable institution, and her duties were arduous and exacting. On Tuesday of each week she was particularly tried on account of the weekly meeting and inspection of a board of lady visitors, and that night was often sleepless. Her eczema of the face and hands was always aggravated on the succeeding morning; indeed, the eruption which would often yield very satisfactorily to treatment during the week, would burst out afresh on the Wednesday morning succeeding the visit. This had been the case for some months before I saw her, and was verified by myself again and again.

A prominent clergyman, aged fifty-two years, had for many years an eczema of the head and face, which was always greatly aggravated on Monday after the Sabbath's mental work and strain. This I myself observed on a number of occasions. The same occurred sometimes during the week, whenever extra work, such as a public address, was called for.

In another case, that of a lady, aged twenty-two years, any nervous excitement or household disturbance and hysterical crying would be followed by a fresh outburst of eczema of the hands.

In numbers of instances I have seen business or legal worry followed shortly by eczema. Mentioning the matter to an exceedingly intelligent gentleman, now aged forty-two years, whom I have seen off and on with eczema for ten years, he remarked on the truth of the observation, and said: "You may quote me as a striking illustration of the effect of nervous strain in producing eczema again and again."

Some knowledge in regard to the effect of nervous strain in the production of eczema may be learned, I think, from a study of the statistics of cases, especially in private practice. In the accompanying tables—I. and II.—are given the sex and age of 5000 cases of eczema under my personal observation, arranged in semi-decades, but time permits only one or two points to be noticed. It will be seen that about one-half of the cases were in males—nearly 54 per cent.; but if the tables be examined more closely, it will be found that among the private patients the males formed a much larger proportion, namely, almost 59 per cent. If now we analyze this more closely, we find that during the period of manhood, from thirty to fifty years of age, when the greatest strain of life comes on men, the number of males in private practice is about double that of females.

TABLE I.

TABLE OF PUBLIC AND PRIVATE ECZEMA CASES.

AGE.	PRIVATE.			PUBLIC.			
	Male.	Female.	Total.	Male	Female	Total.	Grand total.
6 mos. and under	48	32	80	65	62	127	207
6 mos. to 1 year	35	13	48	56	42	98	146
1 year to 2 years	30	30	60	91	47	138	198
2 " 3 "	26	21	47	69	51	120	167
3 " 4 "	11	17	28	49	58	107	135
4 " 5 "	7	10	17	44	18	62	79
5 " 10 "	36	42	78	108	111	219	297
10 " 15 "	22	47	69	53	82	135	204
15 " 20 "	41	73	114	76	102	178	292
20 " 25 "	62	86	148	119	127	246	394
25 " 30 "	92	96	188	135	112	247	435
30 " 35 "	129	66	195	116	84	202	397
35 " 40 "	135	69	204	104	119	223	427
40 " 45 "	124	63	187	76	107	183	370
45 " 50 "	113	67	180	76	121	197	377
50 " 55 "	97	54	151	94	108	202	353
55 " 60 "	90	41	131	64	69	133	264
60 " 65 "	67	31	98	52	49	101	199
65 " 70 "	48	14	62	39	31	70	132
70 " 75 "	34	11	45	31	13	44	89
75 " 80 "	16	6	22	8	5	13	35
80 " 85 "	4	4	8	1	2	3	11
85 " 90 "	2	2	4	...	1	1	5
90 and more	2	...	2	1	...	1	3
	1271	895	2166	1527	1523	3050	5216

Looking now at the column relating to male patients in private practice, we find that between ten and twenty years of age there were but 63 cases, whereas, in the next decade there were 154 cases, or about two and one-half times as many; while during the next decade, that between thirty and forty years of age, there were no less than 264 cases, or more than four times as many cases as during the developmental period—from ten to twenty years—before the cares of life begin to weigh heavily on man. The next decade, that between forty and fifty years, shows also a large number, namely, 237 cases. Thus, between the ages of thirty and fifty, when the nervous strain of life comes most on men, the disease is enormously increased, there being no less than 501

cases, or nearly 40 per cent. of all the instances of eczema in males, in private practice, during this period.

The increased proportion of cases of eczema in males during this period is also forcibly shown in Table II., where the proportion of eczema cases to living people is shown in periods of five years each. Here we see that while the percentage of persons living between the ages of twenty-five and thirty years is only 7.5, the proportion of the eczema cases occurring during this five years is 8.3, and the increased ratio is shown in each semi-decade up to sixty years of age.

TABLE II.

COMPARISON OF PERCENTAGE OF ECZEMA PATIENTS AT DIFFERENT AGES WITH THE PERCENTAGE OF INDIVIDUALS ALIVE AT THOSE AGES, AS SHOWN BY THE CENSUS.

Ages.	Per cent. of ages of all persons.	Per cent. of eczema patients.	Relation between the two.
Under 1 year . .	2.2	6.7	+
1 to 2 years . .	2.0	3.8	+
2 " 3 " . .	1.9	3.2	+
3 " 4 " . .	1.8	2.6	+
4 " 5 " . .	1.8	1.5	—
5 " 10 " . .	8.7	5.7	—
10 " 15 " . .	8.4	3.9	—
15 " 20 " . .	8.2	5 6	—
20 " 25 " . .	7.9	7.6	—
25 " 30 " . .	7.5	8.3	+
30 " 35 " . .	7.2	7.6	+
35 " 40 " . .	6.7	8.2	+
40 " 45 " . .	6.3	7.1	+
45 " 50 " . .	5.9	7.2	+
50 " 55 " . .	5.4	6.8	+
55 " 60 " . .	4.8	5.1	+
60 " 65 " . .	4.1	3.8	—
65 " 70 " . .	3.3	2.5	—
70 " 75 " . .	2.4	1.7	—
75 " 80 " . .	1.5	0.67	—
80 " 85 " . .	0.7	0.21	—
85 " 90 " . .	0.2	0.1	—
90 and over . .	0.07	0.056	—

Exactly how this nerve-strain and nerve-exhaustion operate to induce eczema cannot now be stated with certainty, but, clinically, the fact appears very certain when cases are studied with sufficient exactness. This view is also supported by many writers, and finds corroboration in such expressions as the "nervous debility" of Wilson, and "faulty innervation" of Hebra, Tilbury Fox, and others. It is interesting to note that the elder Hebra[1] expressed himself convinced that "faulty innervation played the principal *rôle* in the production of eczema"; although now so often quoted as a localist, he recognized the internal and general causation of many skin-affections to a much greater extent than is commonly believed. Nikolski[2] has recently reported some cases illustrating the neurotic origin of eczema.

Finally, the therapeutic measures which are of service in many cases of eczema point strongly to a general neurotic causation. Arsenic, strychnine, quinine, preparations of phosphorus and iron, cod-liver oil, and other remedies which are of use in neurotic states, form the basis of much of the internal treatment of eczema; ergot also, as especially shown by Witzel,[1] often proves very valuable, acting, as it is claimed, through the vasomotor nervous system. Electricity likewise, in the form of general galvanization, has been reported on favorably by Beard,[2] Fitch,[3] Morton,[4] Eloy,[5] and others. It is quite possible, also, as suggested by Tilbury Fox,[6] that the beneficial effect of sedative local measures, used early in acute eczema, may be ascribed in part, at least, to a reflex neurotic action.

(To be concluded.)

[1] Hebra: Lehrb. der Hautkrankheiten, Erlangen, 1879, vol. i. p. 763.

[2] Nikolski: Quoted in Viertelj. für Derm. und Syph., vol. xix. p. 1020, 1887.

[1] Witzel: Loc. cit.

[2] Beard: Archives of Dermatology, New York, 1875, p. 325.

[3] Fitch: Ibid., New York, 1875, p. 55.

[4] Morton: New York Medical Record, April 5, 1879, p. 331.

[5] Eloy: l'Union Méd., September 13, 1883; Journ. Cutan. and Ven. Dis., 1884, p. 58.

[6] Fox: Journ. Cutan. Med., iv. p. 170.

A NEW MODE OF EXHIBITING SULPHONAL.

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UNQUESTIONABLY the great disadvantage of sulphonal over other recently-introduced hypnotics is its insolubility and consequent slowness of action. As ordinarily administered, dry upon the tongue, or suspended in mucilage, etc., the result, in most cases, is unsatisfactory. Even after a decided dose hours often elapse before sleep is obtained, and, usually, a condition of very annoying semi-somnolence is maintained throughout the greater part of the subsequent day. Unpleasant effects of this sort are so frequent that many physicians are deterred from prescribing what has proved in my hands the most satisfactory of the newer hypnotics. These effects obtain because of the very slow diffusion of the drug when taken in a state of simple suspension, the whole amount not entering the circulation for many hours after its ingestion.

I desire to call attention to a more satisfactory mode of administering sulphonal; a method that occurred to me when having occasion to take the drug a year or more ago, and one which has subsequently afforded me results in practice not in the least obtainable by its use in the common way. My method is as follows: I direct that just before retiring the sulphonal powder be well stirred in a

glass two-thirds full of boiling water (about six fluid-ounces) until entirely dissolved. The water must be boiling, and to insure that it is at the boiling-point when brought in contact with the sulphonal, it had better be heated on the spot. It can be boiled in a tin-cup over the gas, or over a spirit-lamp. After the sulphonal has entered into solution, which will occur in a moment or two if it be well-stirred, cold water may be very cautiously added to reduce the liquid to a drinkable temperature, which, if the patient is accustomed to taking hot fluids, *will be one not sufficient to cause the slightest precipitation of the drug;* or the hot solution of sulphonal may be permitted to cool to this temperature, the cooling process being facilitated by continued stirring. To insure success the sulphonal must be taken *wholly dissolved*, and the hotter the solution is drunk the better. It is surprising to one accustomed to prescribing this hypnotic in suspension several hours before bed-time, to allow for what has been called the period of therapeutic incubation, to note the prompt and satisfactory result of this simple manœuvre. The hot solution dilates the gastric vessels and stimulates them to rapid absorption, so that diffusion takes place from the stomach probably before slight or any precipitation of the drug occurs; entirely unlike the result that follows when the sulphonal is ingested in a state of simple suspension. In the latter case gastric absorption can scarcely occur, and hours are perhaps consumed before the whole amount taken enters the blood. The period of therapeutic incubation is practically done away with. Sleep results in most cases in a very few moments, and seems to be more profound and dreamless than that from a larger dose taken in the ordinary way, and the annoying condition of drowiness usually present on the subsequent day is scarcely felt if the dose be properly graduated. The hot solution, which has a slightly unpleasant taste from the dissolved sulphonal, may be rendered decidedly palatable by the addition of a tablespoonful of some such *liqueur* as *crême de menthe* ("green mint"), which, apart from its efficiency in this direction, will probably tend to promote still more rapid absorption of the drug. To obtain an immediate and altogether satisfactory result from this method it is desirable that the stomach be empty or at least comparatively free from food, so that precipitation be not favored and absorption delayed by the entanglement of particles of sulphonal and undigested food, but as it is unnecessary to take the dose until the retiring hour there will usually be no difficulty from this.

1231 WALNUT STREET, PHILA.

Obituary.—John Marshall, F.R.C.S., F.R.S., LL.D., the well known English author, anatomist, and physiologist, died January 3, 1891, in London, aged seventy-three years. His death was due to acute bronchitis.

AN EXPERIMENTAL AND CLINICAL STUDY OF ERGOT.[1]

BY JOHN C. HEMMETER, PH. D., M.D.,
OF BALTIMORE, MD.

PART I. *Experimental.*—Our knowledge of the physiological action and therapeutic application of such a complex drug as ergot, must be based upon a perception of relations. In no department of experimental therapeutics do we meet with such manifold and contradictory results, or with more widely-digressing theories than those concerning the action of ergot. This is especially true in the investigations that have hitherto been made relating to the nature and cause of the contraction of the pregnant and non-pregnant uterus, which is produced by this drug. Up to the present time it has not been established whether the action of ergot in this case is a peripheral or central one; that is, whether ergot acts by innervating the uterus through the spinal cord, or directly on the muscular fibre of the organ.

H. C. Wood, in the last edition of his *Treatise on Therapeutics*, admits the uncertainty, but thinks that the drift of present evidence is toward peripheral action. The only evidence which he quotes to base this view upon is that of Boreischal,[2] who in several experiments succeeded in producing violent contractions of the uterus after dividing all of the nervous connections of the organ. Opposed to these results we find those of A. Wernich,[3] who found that no contractions of the unimpregnated womb were produced by ergot after section of the spinal cord. Wernich made only two experiments to decide the point, and any possible force his result might have is destroyed by the fact that he himself does not attribute much importance to it, because the death of his animal took place immediately after the experiment as a result of the operation.

T. Lauder Brunton, in his *Pharmacology and Therapeutics* (1885), intimates that ergot possibly acts like ammonia—producing contraction of the uterus after all nervous connections have been divided—but on the whole gives no experimental evidence for deciding the point in question.

Rosenbach[4] is inclined to accept a direct and local action of ergot upon the muscular tissue of the uterus, indirectly causing contraction by bringing on acute anæmia of the organ.[5] C. Binz holds that as long as we have no better proofs than the present, to believe the opposite we must accept (mainly from clinical observations) that ergot

[1] Experiments were made in the Biological Laboratory of Johns Hopkins University, Baltimore.

[2] Loc. cit., p. 548.

[3] Virchow's Archiv, 1872, B. lvi. S. 510.

[4] Arzneimittellehre.

[5] Pharmakolog. Untersuchungen, Wurzburg, 1879, Bd. iii. S. 78.

directly acts upon the uterus in producing contraction, and not by innervation through the spinal cord.[1] Röhrig,[2] again, is directly opposed to this opinion.

These contradictory statements may be partly explained by the fact that the uteri of different animals vary greatly with regard to their irritability and to the manner in which they respond to stimulations by general contractions.

The uterus of the dog and the cat is not very irritable, and responds only to very strong stimulations, and even then rarely by general contraction. The uterus of the rabbit, however, is very irritable, and at times exhibits spontaneous contractions when no direct stimulus has been employed. Secondary circumstances, such as manipulation incident to the vivisection, opening of the abdominal cavity, and the mere access of the air to the organ are sufficient to cause it to contract. These contractions soon come to a close and then the uterus lies perfectly at rest. It is in these periods of rest that the experiments with stimulation should be undertaken. Schlesinger[3] has called attention to the availability of virgin rabbits for these investigations. In these animals one generally finds the womb completely at rest though responding promptly to applied stimuli. It is not difficult to distinguish the uterus of a virgin animal from one which has borne young. In nine out of the twelve experiments which I made concerning this subject, I made use of so-called virgin rabbits.

The contraction-waves of the uterus generally pass over the muscular layers of the organ in a definite direction; as a rule, they begin as regular peristaltic movements at the abdominal end of the tubes and horns and radiate toward the os, or in rare instances beginning as antiperistaltic movements at the os, they progress over the body of the uterus, shortening it considerably, and run out toward the tubular end of the horns. In addition to these general peristaltic movements, one can notice after circumscribed irritation of the organ, certain local, more or less sharply-defined, circular contractive narrowings or constrictions, which Röhrig, I think, has designated as strictures; these may progress outward over the horns in alternating directions. Finally, by constant increase of the stimulation, the peristaltic contractions as well as the strictures may pass into spasmodic or tetanic contractions under which the uterus may assume an almost erect position. Some of the animals upon which I experimented were pregnant; the uterine contractions in these cases were very intense and lasted from one hour to an hour and a half (whilst in non-pregnant animals they cease in from three to six minutes) when the uterus fell into a state of repose. These periods of repose must always be waited for, before any experiments with stimulation can be undertaken.

A part of the discrepancies in the opinion of the investigators mentioned might be explained by the fact that the quality and efficacy of ergot and of its preparations obtainable in pharmacies vary greatly. This is a misleading circumstance, both in practice and in the laboratory, because no definite chemical tests are known which can be used to establish the therapeutic value of a given sample. In a large number of therapeutic substances we are enabled to test their value by simply determining in them the percentage of definite, crystallizable alkaloids or other principles upon which the therapeutic action of the drugs chiefly depends. In ergot the case is different. Its constituents are numerous, and judging by the widely-differing and often contradictory results obtained by different investigators, are at best, not well defined. This is most likely due to the fact that nearly all the constituents are uncrystallizable, and hence are very difficult to separate without contamination with other extractives soluble in the same menstruum. Nor does the literature on the subject up to the present day enable us to conclude definitely to which of these rather indefinite organic principles the peculiar therapeutic properties of ergot are due. It seems probable from a study of the results of Buchheim, Dragendorff, Zweifel, and recently of Kobert, that at least three of its constituents are of importance. These are sclerotic acid, which Kobert designates as ergotinic acid, scleromucin, and the cornutin of the latter investigator, who has recently called our attention to a new body in ergot, namely, sphacelinic acid. It is also established that the characteristic physiological effects of the drug are not due to either one of these bodies, but to a combination of them. Thus ergotinic acid produces contraction of the arterioles, but not of the muscular fibres of the uterus. According to Kobert, the constituents which produce contraction of this organ are cornutin and sphacelinic acid,[1] but the whole subject of the active ingredients still remains enveloped in much uncertainty.

As the watery infusion of fresh ergot (this having been used by former investigators) was found to undergo changes very rapidly, I concluded to resort to the fluid extract of ergot, but found this as obtained from various sources very variable, both in physical properties and therapeutic efficacy, some of the specimens having a very offensive odor. In

[1] Vorlesungen über Pharmacologie, Bd. xi. S. 298.

[2] Arch. f. Pathol. Anat., Bd. lxxvi. S. 23.

[3] Uber die Centra der Gefäss und Uterus nerven, Medic. Jahrbücher, 1874, Heft 1.

[1] See also Buchheim, Archiv. f. exper. Path. u. Pharmak, 1874, Bd. iii. S. 1. Zweifel, ibid. Bd. iv. S. 387, and Dragendorff u. Podwissotzky, ibid. Bd. vi. S. 153.

two German preparations of ergotin and one liquid ergotin prepared in Basle, Switzerland, and specially recommended by the manufacturer for hypodermic use, a very unpleasant fœtid odor reminding of decomposed organic matter was noticeable, and the last-named used hypodermically proved very irritating, causing an abscess in a patient suffering from goitre.

Some of the secondary results in the experiments upon animals, which I shall presently describe, were undoubtedly caused by the impurities of the ergot used. (Emboli in the pulmonary and cardiac circulation.) My attention was at last called to a form of liquid ergotin made in Baltimore by Sharp & Dohme, which gave evidences of being a standard preparation, both in clinical and experimental application. I have had some of this ergot in my possession for nearly ten months (since February 3, 1890). It has deposited no sediment, has a fresh, pure odor, and is very effective. This ergotin solution, which is probably the most concentrated liquid preparation of ergot that can be obtained, has since become known under the name of ergotole, to distinguish it from the numerous and widely-differing preparations sold under the name of ergotin. In the experiments to be described this form of ergot was used, together with two forms of fluid extract of ergot, the officinal containing hydrochloric acid, the other no acid, which, however, were more bulky.

After the injection of 1 c.c. of fluid extract of ergot (about 16 minims), the capillaries and arterioles in the omentum of a large rabbit could be seen to contract within from five to eight minutes, and the uterus showed peristaltic contractions. On the other hand, 0.25 c.c. (4 minims) of the liquid ergotin just described produced the same effect on the arterioles in from two to three minutes (rarely five minutes), and the contractions of the uterus were more active and followed each other with greater rapidity. The ergot in both cases was diluted with the same quantity of distilled water, and injected into the jugular vein in preference to the hypodermic method, as the absorption of solutions of ergot from beneath the skin in animals is very slow. This does not seem to be the case in the human being. The rabbit omentum or mesentery was gently spread over a warm stage, and constantly kept under warm normal salt solution; the microscopical examination was made with a Zeiss objective D, ocular A. The irritation was applied in the form of the constant and also the interrupted current, but chiefly the latter, by making use of an induction coil. The time elapsing between stimulation and contraction was measured by a second metronome and at times by a watch. The electric stimulations referred to were employed to test the state of excitability of the uterus. The objects of the experiments which will now be described were:

1. To determine whether the contractions of the uterus by ergot is of centric or peripheral origin.

2. Whether the peristalsis of the intestines is increased or diminished by ergot. If increased, whether this be due to a centric or peripheral action of ergot.

3. Whether the cause of the contraction of the bloodvessels in the omentum is central or peripheral.

4. Whether ergot produces a rise or a fall of blood-pressure. Whatever change occurs, is it due to an action on the heart and arteries or on the spinal cord.

5. The action of ergot on temperature.

First. We will first take up the question of the centric or peripheral origin of the contractions of the uterus. As both chloroform and ether reduce the peristaltic contractions of the uterus in animals, the rabbits used for this purpose were curarized and given, in some cases, a rectal injection of morphine. The curare must be injected in very small quantities, otherwise it will also affect the uterine contractions.

It was necessary in order to solve this problem to isolate the uterus from all nervous connection and then observe whether it would contract after injecting ergot into the jugular vein. It has been established beyond a doubt by Goltz[1] that the centre for innervation of the uterus is located in the lumbar region of the cord. For after cutting the cord at the first lumbar vertebra in a female dog, nine months old, and allowing the wound to heal, the animal in course of time became pregnant and brought forth normal healthy pups. This experiment proves, it is true, that the lumbar cord is sufficient to innervate all these processes, but not that there are no centres in the cord above the line of the first lumbar vertebra, which may also have some control over pregnancy and parturition. The experiments of Röhrig to which I have referred, show conclusively that stimulation of the upper end of the cord after it has been divided at the first lumbar vertebra (according to Goltz's method) still produces distinct muscular contraction in the uterus. This seems confirmatory of Th. Kömer's results,[2] according to whom the sympathetic nerves which leave the cord at the level of the eleventh and twelfth dorsal vertebræ go to the uterus. This nerve track was, therefore, not cut through in Goltz's experiments, and to isolate the uterus completely from all nervous connections we would have to destroy the cord from the tenth dorsal vertebra down to the sacrum, or cut through all nerves which run from the cord to the organ. We decided

[1] Ueber die Functionen des Lendenmarks des Hundes. Pflüger's Archiv, Bd. viii.

[2] Studien des Physiol. Instituts zu Breslau, Heft 3.

upon destruction of the cord, since intersection of the numerous nerve-fibres going to the uterus is attended with much manipulation and exposure of the abdominal organs, besides being a matter of considerable difficulty. The cord was accordingly destroyed by opening it at the tenth dorsal vertebra and running a white-hot copper wire down through the vertebral canal to the cauda equina. This procedure is not difficult and its immediate results do not injure the vital powers of the animal.

Schlesinger[1] has shown that the venous condition of the blood which is caused by temporary interruption of respiration acts as a stimulant to general contractions of the uterus. Röhrig[2] has confirmed this result, and shown at the same time that the contractions do not occur after the destruction of the cord. Spiegelberg[3] has shown that compression of the aorta, cutting off arterial blood-supply, also, in a few seconds produces contraction of the uterus. This has been confirmed by Röhrig and Schlesinger (*loc. cit.*).

Before destroying the cord in the manner stated, we tested the reaction of the uteri of the rabbits by temporary suspension of respiration, by compression of the aorta, and by electricity. After we had assured ourselves that contractions promptly occurred in from six to ten seconds after respiration was suspended (a rabbit breathes 40 in a minute), and in from ten to twenty-five seconds after compression of the aorta, and immediately following electrical stimulation, ergot was injected into a canula in the jugular vein in the form of 0.25 c.c. (4 minims) of ergotole dissolved in 1 c.c. (16 minims) of defibrinated blood from another rabbit or in the same quantity of normal salt solution. Uterine contractions began to pass gently over the organs four minutes after the injection, gradually becoming more intense. When 0.5 c.c. of ergotole was injected, or 1 c.c. (16 minims) of the fluid extract, the contractions of the uteri became tetanic in from three to five minutes after the injection, and continued to increase in violence for thirty minutes (average), then gradually decreased until they ceased altogether, in from fifty to sixty-three minutes after the first injection. When the contractions had thus passed off, and the uteri resumed a state of complete rest, the movements could be reproduced by a second jugular injection of ergotole. To reproduce the same effect the quantity as a rule had to be doubled; the tetanus then came on sooner, but lasted only twenty minutes, when the effects of the second injection ceased also.

When uterine tetanus is produced by ergot there is no relaxation between the spasms, as in the case of contractions produced by compression of the aorta or by suspension of respiration.

When the physiological effect of ergot had thus been established with a single injection of 4 minims of ergotole, and the organ had resumed a position of rest, the cord was destroyed from the tenth dorsal vertebra down to the cauda equina by means of a hot wire.

A second injection of ergotole (1 c.c. or 16 minims) into the jugular vein produces no contraction of the uterus. The result of a third injection of 32 minims (2 c.c.) of ergotole is also negative. Suspension of respiration and compression of the aorta for one minute each time now exert no effect upon the organ.

Electric stimulation, however, is promptly followed by energetic contractions. Injections into the jugular vein of three-per-cent. solutions of ammonia in defibrinated blood (1 c.c. of three-per-cent. solution in NH_3 in 2 c.c. defibrinated blood) are followed by strong uterine contractions.

The following is a tabulated account of one of the experiments:

October 27, 1890. A large, non-pregnant, albino rabbit is subjected to tracheotomy and curarized. A canula, with a rubber-tube attached to it, is tied into the cardiac end of the jugular vein, the other end of the rubber canula is filled with defibrinated rabbit's blood and clamped. Uterus exposed by abdominal section.

Time.	Kind of irritant or manipulation.	Result.
3.10 P. M.	....	Uterus at rest.
3.12	Faradic stimulation, positive pole on lumbar cord, negative on uterus, distance of coil 80 mm.	Strong contractions immediately.
3.15.30	Compression of aorta.	
3 16	" " "	Strong contractions.
3.17		
3.18	....	Uterus at rest.
3.18.40	Suspension of respiration.	
3.20	....	Strong peristaltic waves.
3.20.30	Respiration resumed.	Peristalsis continues.
3.22	....	Peristalsis ceases.
3.23 30	....	Uterus at rest.
3.25	Injection 25 c.c. ergotole.	" "
3.26	....	" "
3.27	....	" "
3.29	....	Slight undulations over horns.
3.30	....	Antiperistaltic movements marked.
3.31	....	General contractions of uterus.
3.33	....	General peristalsis and antiperistalsis occur once in 15 to 20 seconds.
3.34	....	They occur over the entire organ.
3.40	....	General peristaltic movements occur every ten seconds.

[1] Medicin. Jahrb., 1873, Bd. 1.
[2] Virchow's Archiv, Bd. 76, S. 12.
[3] Zeitschrift für rationelle Medicine, 3 Reihe, Bd. ii.

Time.	Kind of irritant or manipulation.	Result.
3.45	.....	Contractions of intensity occur all over uterus about four times in a minute.
3.50	.....	Contractions less intense, and but two in a minute.
3.52.30	.....	One contraction in a minute.
3.54	.....	Uterus at rest.
3.55	.....	" "
3.56	.....	" "
3.57	Injection 5 c.c. ergotole.	" "
3.58	.....	" "
4.0	.....	" "
4.2	.....	Slight waves of contraction along both cornua.
4.3	.....	Contractions over the body of the uterus.
4.5	.....	General peristalsis and antiperistalsis.
4.6	.....	Marked and intense general contractions every 10 seconds.
4.7	.....	Contractions following each other in rapid succession.
4.10	.....	Contractions occur every 15 seconds.
4.15	.....	Muscular spasms about four times a minute.
4.20	.....	Contractions only once in 30 seconds.
4.22	.....	Contractions ceasing.
4.25	.....	Uterus at rest.
4.26	.....	" "
4.27	.....	" "
4.28	Destruction of cord from 10th dorsal vertebra.	" "
4.30	Injection 5 c.c. ergotole.	" "
4.35	.....	No result; uterus at rest.
4.36	.....	" " " "
4.38	Injection 5 c.c. ergotole.	" " " "
4.40	.....	" " " "
4.45	.....	" " " "
4.48	Stimulation with faradic current.	Immediate and rapid contractions.
4.50	Injection 1 c.c. 3 per. ct. solution of ammonia.	
4.51	.....	Forcible antiperistalsis.
5.0	.....	The peristalsis produced by the ammonia lasted for 15 minutes, and continued for 5 minutes after the uterus was cut out of the body.

The foregoing table is an account of one of twelve successful experiments which convince us that (1) injections of from 0.25 c.c. to 0.5 c.c. of ergotole, or of 1 c.c. to 1.5 c.c. fluid extract of secale cornutum, will produce intense uterine contractions if inserted into the jugular vein when the cord is intact; (2) that when the cord is destroyed by a hot wire from the tenth dorsal vertebra downward, the above injections, and even double the quantity, have no effect; (3) that electric stimulation will cause prompt uterine contractions even after destruction of the cord; (4) that injections of 1 c.c. of a three-per-cent. solution of ammonium hydroxide will cause uterine contractions after destruction of the cord.

The conclusion is that ergot in producing contractions of the uterus acts primarily and essentially upon the lumbar cord, *i.e.*, its action in causing peristalsis of the uterus is centric, not peripheral.

In three of the experiments it was plainly shown that compression of the aorta and suspension of respiration do not cause uterine contractions after the cord is destroyed, therefore the action of the changes caused by these procedures are also centric and not peripheral. A three-per-cent. solution of ammonia was injected at the end of each experiment, when ergot no longer produced any contractions after cord destruction, and the prompt peristalsis occurring after the injection of ammonia under these conditions proves its action to be exerted directly upon the uterus and to be peripheral.

Does ergot cause increased intestinal peristalsis by direct local action or by exerting an influence upon the spinal cord?

This question could be conveniently solved together with the preceding one, as the same experimental method was applicable to both inquiries. Suffice it to say that in the twelve experiments upon the action of ergot on the uterus, careful observations were made of the peristalsis of the intestines. There is already much good evidence to show that ergot does cause increased intestinal peristalsis. Mr. Samuel A. Wright,[1] in a series of experiments, found very active peristalsis when ergot had been given by the mouth. Dr. Wernich[2] noticed violent peristaltic movements of the intestines following the injection of ergot. This was observed in rabbits whose abdomens were opened; in some cases, however, even through the uninjured abdominal walls. Wernich's statements have been confirmed by Handelin.[3]

Basch and Mayer[4] have stated that the dyspnœic condition of the blood caused by suspension of respiration stimulates not only uterine but also intestinal peristalsis. We are enabled to confirm this observation and to add that compression of the aorta also increases intestinal peristalsis. This compression was in two cases exerted upon the thoracic aorta to avoid direct mechanical irritation of the intestines. When the abdomen of a healthy rabbit is opened, the intestines being kept under normal salt solution, and allowed to remain undisturbed, peristaltic movements will cease in about from eight to ten minutes. If after this absence of motion is established, 0.5 c.c. of ergotole, or 1 c.c. of fluid extract of ergot, is injected with twice the volume (2 c.c.) of normal salt solution, there is at first no change observable in the intestines, but in from six to eight minutes (about the time the uterus shows signs of contraction) a fine quivering is plainly

[1] Edinburgh Med. and Surg. Journ., Oct. 1839, vol. iii.
[2] Virchow's Archiv, 1872, Bd. lvi. S. 515.
[3] Schmidt's Jahrbücher, Bd. clv.
[4] Quoted by Röhrig, Virchow's Archiv, Bd. lxxv.

noticeable along those parts of the ileum which are most easily visible.

In ten minutes distinct wavy contractions are seen to traverse the ileum and the colon. With a little care it can be proven that lumps of half-digested corn and cabbage are forced along the duodenum. By help of these masses of food it is not difficult to watch the progress of the peristalsis, which continues even after the motions of the uterus cease. In our experience the intestinal peristalsis kept up for forty-eight minutes, or nearly twenty minutes longer than the action of the uterus. When the peristalsis of the intestines caused by the first injection of ergot ceases, it can be reproduced by a second injection, which was generally allowed to run into the jugular vein from a eudiometer. If the cord was destroyed from the tenth dorsal vertebra down to the cauda equina, it was in no case possible to produce intestinal peristalsis by twice or even three times the quantity of ergot which had been previously used. It is justifiable, therefore, to conclude that ergot, in producing intestinal peristalsis, acts directly on the cord and only reflexly upon the intestines, its action in this case, too, being centric, not peripheral.

Contraction of arterioles under ergot.—We have made observations on this subject by microscopical examination of the frog's web and the rabbit's omentum and mesentery.

According to H. C. Wood, as early as 1827 M. Courhant advanced the opinion that ergot produces a spasm of the bloodvessels. In 1870 Dr. C. L. Holmes[1] published some observations on this point, and states that when the bloodvessels in the frog's web are watched under the microscope and the animal is poisoned with ergot, they can be seen to undergo a great contraction. A. Wernich[2] reports that when animals in which the arteries of the thigh, back, pia mater, etc., were laid bare, received full doses of ergot, these arteries could be distinctly seen to undergo decided contractions. Paul Vogt has confirmed these observations, also S. Kersch,[3] Boldt,[4] and Max Muller.[5] Boldt asserts that a wave-like peristalsis ran through the capillaries in ergotized animals. Peton, in 1878, observed in a rabbit's ear a complete change of color, from red to pale, under ergot, and proved that this anæmiation was independent of any influence exerted by the central nervous system. The following observation of Dr. Paul Vogt[6] is very interesting: He states that the dilated vessels of the ear of a rabbit whose cervical ganglion had been extirpated could not be made to contract by the hypodermic injection of ergot. P. Eberty's results[1] agree with those of Vogt. On the other hand, Dr. Holmes[2] asserts that after section of the sympathetic nerve and injection of the ergot, the vessels supplied by the divided nerve can be seen to contract, and Wernich[3] confirms this observation. Vogt's and Eberty's experiments, therefore, point to a centric origin of the contraction of the arteries, and those of Holmes and Wernich to a peripheric action. H. C. Wood[4] has especially investigated this subject, but chiefly with regard to the origin of the rise of arterial pressure, which will be most appropriately be considered under the next heading.

Although we examined the omentums of six of the rabbits on which we had made observations upon the uterus, we made six further experiments on this subject of arterial contraction under ergot. The omentum was constantly kept under a thin, slow current of warm normal salt solution.

As H. C. Wood says, that judging by the eye as to whether a vessel does or does not contract is very unreliable, we made use of the micrometer eyepiece and also of the so-called jury system. In this method the experimenter calls in the help of as many fellow-workers as are available, and directing them to watch with the micrometer a certain network of vessels in the omentum, without letting them see his injection of ergot into the jugular vein, asks them, "Tell me as soon as you notice any difference in the size of those vessels—do they grow larger or smaller?" Of ten observers assisting in this manner nine answered "smaller" within from two to three minutes after 0.5 c.c. of fluid extract of secale cornutum, or 0.25 c.c. of ergotole was injected. The tenth man said he was unable to tell, but thought the vessels remained about the same size.

It is but a fair conclusion in this case that ergot injected into the jugular vein produces contraction of the arterioles and capillaries in the omentum of rabbits with the spinal cord intact. The same study was made on the vessels of the ears of the rabbits, and especially on the web of the frog's foot, with the result that of all the ten observers the answer was the same, to the effect that a contraction had taken place when the ergot was allowed to run into the jugular vein from a eudiometer, but unseen by them. In animals, however, in which the spinal cord had been destroyed from the cervical region to the cauda, the vagi divided and artificial res-

[1] Archives de Physiol., t. iii.
[2] Virchow's Archiv, 1872, Bd. lvi. S. 510.
[3] Unfortunately it was impossible to have access to Dr. Kersch's paper. There is an abstract of it in Schmidt's Jahrbücher, Bd. clx. S. 120.
[4] Schmidt's Jahrbücher, March, 1872.
[5] Berlin. klin. Wochenschrift, 1874, p. 305.
[6] Ibid., 1869, No. xii.

[1] Inaug. Dissert., Halle, 1873.
[2] Loc. cit.
[3] Loc. cit.
[4] Phila. Med. Times, vol. iv.

piration set up, no contraction of the arterioles and capillaries was observable after ergot had been injected. Here the vote again stood 9 to 1 in favor of no reduction in the size of the vessels, the same man again declaring that he was not able to decide. This observation was also made upon the ears of the rabbits.

The conclusion is that ergot produces constriction of the arterioles and capillaries in the omentum and ear of rabbits and in the frog's web as long as the cord and the vagi are intact. These being destroyed constriction is no longer produced by the drug; its action in this case is centric, not peripheral.

The following study merits consideration in this connection: By means of a hot wire an inflammatory process was set up in one portion of the omentum. The congestion and infiltration extended around the point of irritation, making an area of inflamed tissue the size of a silver dollar. Whilst this area was under consideration it was steadily spreading and new capillaries seemed to grow out where three minutes before none had been visible. At this stage ergot was injected into the jugular vein of the animal (rabbit). Within three minutes the congestion and hyperæmia were visibly diminishing, and capillaries which we had observed developing, actually disappeared. We presume that there was no real new development of capillaries during the first hyperæmia, but that such as were already present but invisible because of their minute size became apparent from the general congestion, and disappeared again under the arterial contraction and consequent lessening of blood-supply to the capillaries caused by ergot.

Observations like those of Patrick Nicol and J. Mossop,[1] who noted with the ophthalmoscope the contraction of the retinal vessels after the exhibition of ergot in man, are very suggestive and convincing. I have made the same study of the retinæ of two epileptics at Bay View Asylum. The diminution of the calibre of the vessels under ergot was very apparent. It is not a reliable method, to judge of the effects of a drug in contracting the blood-vessels by observing the capillaries, because the size of these vessels is very variable at all times. Capillaries will not always have the same size in the same region of the body. One such minute vessel may appear so narrow that a single red blood-corpuscle passes through it with difficulty; at another time the same capillary will be wide enough to afford room for two or three such corpuscles to travel abreast. At certain moments, as when the access on the arterial side is blocked, or when blood for some reason ceases to flow into it, the capillary may be empty, its walls collapsed and in contact, and its lumen nearly abolished. At another time, when blood is flowing into it at high pressure, the capillary may be widely distended. It is, therefore, more accurate to watch the arterioles and larger capillaries only, and to base opinion chiefly upon micrometric measurements of the vessels, which is an easier method of observation.

(To be concluded.)

[1] British and Foreign Medico-Chirurg. Rev., vol. i. 1872.

MEDICAL PROGRESS.

Treatment of Nasal Diphtheria.—In the *Revue Générale de Clinique et de Thérapeutique*, RAULIN recommends the following treatment for nasal diphtheria, both in adults and children. The membrane is not to be removed by direct manipulation, but is to be detached by means of antiseptic irrigation, while to prevent its re-formation, the surfaces upon which it is growing are to be covered by the following:

℞.—Lactic acid	. . . .	30 grains.
Carbolic acid	. . . .	45 "
Pure glycerin	. . . .	1 ounce.

After the disappearance of the exudate the catarrhal rhinitis which follows is best treated by means of astringent applications, and by washing out the nostrils with a spray composed of a solution of borate of sodium, or Dobell's solution may be used instead.

The Treatment of Strangulated Hernia by Belladonna and Atropine.—HAGEN, in the *Centralblatt für Chirurgie*, states that in twenty cases of strangulated hernia he has employed the following treatment: For several hours the hernial tumor is carefully treated by inunctions of an ointment made by adding 1 drachm of the extract of belladonna to 10 drachms of benzoated lard, and finally, under the skin, immediately over the tumor, is injected by means of a hypodermic syringe, $\frac{1}{100}$ grain of sulphate of atropine, which may be repeated if necessary. At the same time with these inunctions and injections taxis is employed, and in a certain number of cases Hagen found it unnecessary to resort to herniotomy. The employment of the belladonna and atropine renders the tumor more soft and yielding and furthers spontaneous reduction of tension. The reason for this treatment rests upon the statement of Schmiedeberg that belladonna and atropine in minute doses cause a contraction of the mesenteric vessels and an increased peristalsis of the intestinal walls.

Indications for Operation in Gastric Ulcer.—NISSEN (*St. Petersburger medicinische Wochenschrift*, No. 41, 1890) discusses the question of operative interference in cases of circular ulcer of the stomach, and relates a case from the clinic of Professor Unverricht. The patient was a man, aged forty-six years, who had had the usual signs of an ulcer of the stomach since July, 1887. His disease progressed with the usual symptoms of violent pain in the epigastrium, vomiting of blood, and progressive wasting, until September, 1889, when the symptoms pointed to perforation of the ulcer with commencing peritonitis. Professor Wahl thereupon opened the ab-

domen by an incision in the linea alba between the umbilicus and ensiform cartilage. There was no free gas in the peritoneal sac, and the stomach was easily made to present itself in the wound. Neither palpation nor inspection showed anything abnormal, and the serous coat was smooth and shining, although abnormally vascular. There was no peritonitis, however, and nothing to explain the clinical signs. The abdomen was closed, and the patient progressed fairly well for some days, but a month after the operation his temperature rose, and there was pain in the abdomen, culminating in collapse, from which he died. The clinical diagnosis was that of a perforating ulcer of the stomach with consecutive peritonitis. An examination was made and the left lobe of the liver was ascertained to be adherent to the stomach, and when the adhesions were broken, a small perforation was found in the anterior wall of the stomach. This hole had allowed the contents to escape into the peritoneal cavity. The stomach itself was hour-glass shaped, and had a large ulcer at its lesser curve extending down the anterior wall, where it had given way. In commenting upon this case Nissen points out that the indications for performing an operation, namely, perforation and commencing peritonitis, may not be fulfilled unless the lesser omentum be torn through and the posterior surface of the stomach explored with the finger. Nevertheless, should an ulcer be situated there, adhesions with the pancreas will often be found. A better means of diagnosing the position of ulcers seems essential to the successful performance of these operations.—*British Medical Journal*, January 3, 1891.

Treatment of Chancroid.—In *La Tribune Médicale*, CASTEL gives a summary of the present treatment of chanchroid. After stating that a large number of remedies have been brought forward during the last few years for the cure of this condition, he points out that iodoform is probably the best of these, and gives the following ways in which iodoform may be employed:

℞.—Iodoform . . . . 2 parts,
Powdered coffee . . . 1 part;

℞.—Iodoform . . . . 10 parts,
Carbolic acid . . . . 1 part;

℞.—Iodoform . . . . 2½ drachms,
Carbolic acid . . . . 2 grains,
Essence of peppermint . 1 to 2 drops;

℞.—Iodoform . . . . ½ ounce,
Camphor . . . . 75 grains,
Essence of roses . . . 2 drops;

℞.—Iodoform . . . . 1 drachm,
Coumarine (principle found in Tonka bean) . . . } 15 grains;

℞.—Iodoform,
Powdered quinine,
Powdered benzoin,
Carbonate of magnesia (saturated with oil of eucalyptus), equal parts of each.

All of these methods mask, to a great extent, the disagreeable odor of the iodoform without in any way interfering with its favorable therapeutic effect. Castel also notes the following formulæ, which have been recommended in America, Germany, and France:

℞.—Salicylic acid . . . . 15 grains,
Powdered chalk or starch . 2 drachms;

℞.—Salicylic acid . . . . 20 grains,
Alcohol . . . . . 45 drops,
Benzoated lard . . . 2 ounces;

℞.—Salicylic acid . . . . 15 grains,
Alcohol . . . . . 2 ounces;

℞.—Salicylic acid . . . . 30 grains,
Alcohol . . . . . 3 ounces.

It is also stated by Castel that pyrogallic acid is a very useful application, and that Terrillon uses the following formula:

℞.—Starch . . . . . 1½ ounces.
Vaseline . . . . . 6 "
Pyrogallic acid . . . 1½ "

The salicylic and pyrogallic acid prescriptions are, of course, very much more powerful stimulants and irritants of the sore than are those of iodoform which have just been given.

The Treatment of Urticaria.—QUINQUAND recommends the following treatment for this troublesome affection: Internally he prescribes the alkalies, the arsenite of sodium, and naphthol; and when the itching becomes intolerable, he relieves it by the following lotion:

℞.—Boric acid . . . . 1½ ounces.
Chloral . . . . . 1 drachm.
Water . . . . . 6 ounces.

The following powder is also of value:

℞.—Powdered starch . . . 1½ ounces.
Oxide of zinc . . . . 3 drachms.
Salicylic acid . . . . 1 drachm.

External Application of Aristol.—The *American Journal of Pharmacy* quotes the following formula for an aristol paste:

℞.—Aristol . . . . . 3 parts.
Alcohol . . . . . 5 "
Ether . . . . . 5 "
Soft soap . . . . 30 " —M.

Soft paraffin is also a good vehicle, and a mixture of equal parts of lanolin and petrolatum has been recommended for the same purpose.

Sulphur in the Treatment of Smallpox.—According to the Paris correspondent of the *Archives of Pediatrics*, DR. ISCAR prescribes sulphur in cases of smallpox, and claims much success. He thinks that it is eliminated by the skin and that this accounts for the good effects. For children he uses the following formula:

℞.—Washed sulphur . . . 2½ drachms.
Glycerin } of each 15 "
Orange-flower water }
Syrup . . . . . 7½ " —M.
Dose, one teaspoonful every hour.

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SATURDAY, JANUARY 31, 1891.

OPPOSITION OF HOMŒOPATHS AND ECLECTICS TO THE MEDICAL EXAMINERS' BILL.

THE MEDICAL NEWS recently published and made editorial comment upon the Bill prepared by the Medical Society of the State of Pennsylvania to establish a State Board of Medical Examiners, similar to those existing in Minnesota, Virginia, and other States. It was then said that the Bill was a just and fair one to the State, the profession, the candidate desiring license to practise, and to those practitioners believing in special dogmas. An opposition to a State Board of Examiners has been developed, however, on the part of the homœopaths and eclectics, who, in combination, have prepared and introduced a Bill into the House of Representatives at Harrisburg, entitled "An Act to establish a State Board of Medical Education," etc. The text of this Bill is published in the present issue of this journal, in order that its provisions may be carefully studied. Its fundamental principle is that the Board shall regulate the extent and character of the preliminary education of intending medical students, and fix the character and time of instruction given in medical schools. It shall issue, without examination, certificates to all graduates of the medical schools of Pennsylvania, which must conform to the standard set by the Board; and shall examine graduates of schools situated outside of Pennsylvania which do not appear to conform to the said standard.

The bill introduced by the homœopathists and eclectics, however, does not seem to be carefully worded or justly conceived. There is no provision to assure the public that the graduating examination in the medical colleges shall be a sufficiently rigid one. "The methods of instruction employed and the facilities for teaching" are to be inspected (see Section 6); but after such inspection the colleges may graduate the most ignorant persons after secret examinations of a most trivial kind. The preliminary examination is also to be regulated as to extent and character, but no attempt is made to see that it is faithfully conducted. The casual reader will see that no protection against medical ignorance and incompetence would be given by such a law. At the present time there are colleges in all parts of the world which have good methods and excellent facilities, but the graduates need not, and often do not, attain much proficiency, because the final examination is too lax.

The defective protection afforded by the bill shows that it is far inferior to the bill to "Establish a State Board of Examiners," published in THE MEDICAL NEWS, January 10, 1891. In addition, the evident intentional injustice of the homœopathic and eclectic Bill to Establish a Board of Medical Education makes its defeat an imperative necessity on the part of the regular medical profession. It is drawn (see Section 1) in such a manner that the appointing Governor must always put the regular profession in a perpetual minority. There must forever be out of the 9 members 6 irregular practitioners upon the Board, viz., 3 homœopaths and 3 eclectics. In other words, the homœopathic and eclectic physicians of Pennsylvania, who number about 1000, are to have police supervision over and power to annul the charters (see Section 8) of the five regular medical colleges of the State, which represent nearly 7000 resident practitioners of rational medicine, and this in face of the fact that the irregular physicians have but a single college in the Commonwealth. Other defects in the law are that professors in medical schools may be appointed on the Board, and that no two members shall be residents of the same county. Consequently, the sole member from Philadelphia County would probably always be a member of the faculty of one of the medical schools. The possibility of just educa-

tional supervision under such circumstances is very doubtful.

It is astonishing that the homœopathic physicians who have long expressed a desire for a higher standard of education, should have combined with the eclectics to defeat the non-sectarian and carefully-drawn bill to create a Board of Medical Examiners. This bill provides that the Governor shall appoint three eclectics from nominees made by the Eclectic Medical Society of Pennsylvania.

We would urge every member of the profession in this State to write to his representatives at Harrisburg, insisting that the basis of representation shall be as fairly proportioned in this act as in any other measure which may become a law. Until we have proper methods of protecting our fellow-citizens and ourselves the profession of Pennsylvania must always be under a cloud of illiteracy and quackery.

CORRESPONDENCE.

PARIS.

Koch's Remedy in Paris.

KOCH'S lymph has now been experimented with in Paris for more than a month. The observations have been made upon medical, dermatological, and surgical cases. In medical cases, such as pulmonary tuberculosis, the results have not been as favorable as was expected. In cutaneous tuberculosis, improvement or changes in the preëxisting condition have certainly been brought about, and seem to indicate that the remedy will prove to be of some value in such diseases. In surgical tuberculosis the results have not been encouraging, but we shall have to wait for months before a positive opinion can be expressed as to the value of the remedy in all classes of tubercular cases.

As soon as the precious lymph was received it was placed in the hands of one of our most careful scientific men, Professor Cornil, the cases treated being watched by his very careful assistants, Professor Chantemesse and Dr. Vidal. Dr. Cornil began his inoculations in the latter part of November. The first patient to receive the treatment was a very extensive case of lupus, covering the whole face and the left arm, forearm, and hand. The often-described action of the remedy was very marked in this patient. Six hours after the injection she began to cough, and it was found that she had tuberculous lesions in the upper portion of her lungs. The second case was one of tubercular epididymitis; the right spermatic cord was about one-half inch thick, while in the left epididymis there was an almost imperceptible nodule. Under the influence of the remedy the right epididymis enlarged and the nodule in the left epididymis became of the size of a hazelnut. No marked general symptoms occurred in this case after the first injection, but after the second injection the patient complained of severe renal pain, and was found to be suffering from hæmaturia and albuminuria, the albumin and blood being present for several days. A third case of lupus was also injected, and while no general symptoms appeared, the local symptoms were pronounced.

Professor Cornil has found that reaction is always produced by the lymph in cases of undoubted tuberculosis. Certain irregularities, however, have been observed, some patients having a very slight rise of temperature, although there was a very marked local reaction in the lung. In some cases both the local and general reaction were marked, while in others the general reaction was marked, although the local was very feeble. The general symptoms differ acccording to the quantity of liquid injected and also the idiosyncrasy of the patient. In a case of tubercular arthritis of the elbow of twenty years' duration which was thought to be cured, five milligrammes of the lymph brought on no reaction. In another case of old tubercular arthritis, no reaction was observed. Professor Cornil concludes that in old tubercular arthritis which has reached the fibrous stage, no reaction will occur. On the contrary, the reaction is much more intense when we have to deal with a rapidly developing recent tuberculosis.

As regards the sensitiveness to the drug, Dr. Cornil presented a patient suffering from tuberculous lesions of the skin, cold abscesses, tuberculous disease of the elbow and wrist, necrosis of the malar bone and pulmonary tuberculosis in the first stage. After the injection, the patient had a red and painful swelling on one finger. It is probable that this tubercular lesion was merely made evident by the injection.

A child about thirteen years old, who had had pulmonary tuberculosis for only a month presented, when admitted, a few dry crackling râles under the left clavicle and also slight dulness on percussion. At the first injection the physical signs were greatly increased.

When one has to deal with latent tuberculosis, the injections increase the physical signs, and render distinct symptoms which were previously doubtful.

All the organs of the body are liable to be affected by Koch's lymph, and two of the lupus patients who were injected, presented marked hæmaturia. The urine, which was of a rose color on the day following the injection, became brown for four or five days after, and contained albumin. On microscopical examination besides free blood-corpuscles, hyaline casts filled with deformed blood-corpuscles, were found.

Three hypotheses are to be considered as an explanation of these phenomena, first, tubercular granulations may have been present in the kidney; second, there may have been a slight nephritis with an epithelial degeneration or an interstitial periglomerulus lesion; or third, these patients may have had amyloid degeneration of the kidneys. As soon as albumin is detected in the urine of a patient treated by these injections, no further injections are given until the urine has returned to normal. Strange to say, in a case of advanced phthisis, with diabetes and a double calculous pyelonephritis, injections instead of bringing on hæmaturia, have shown no action on the kidney—the quantity of pus in the urine has even diminished.

Five nephritic patients who presented skin-lesions were also inoculated, for the purpose of ascertaining the

action of the remedy on them, each receiving one milligramme, but none of them presented any reaction.

As regards temperature, Professor Cornil and Dr. Vidal have observed that the rise is not confined to one day, but may return on several successive days. In one case the temperature reached its maximum in from eight to twelve hours, it then rapidly fell within the four following hours to nearly normal, and in twelve hours again rose to the former height. In another case, one of lupus, the temperature rose daily for three days.

A third case, a girl aged seven years, who received one-half milligramme of the lymph, presented no general reaction immediately after the injection, but between the second and fourth days the temperature rose and varied between 100° F. and 104°. This case shows that one cannot safely make an injection every day.

Dr. Henague has also made examinations of the amount of oxyhæmoglobin in inoculated patients. Out of twenty-two patients he found that in thirteen the oxyhæmoglobin was diminished. In three there was first a diminution then an increase. In three an increase, and in three no variations were observed. A diminution in the quantity of oxyhæmoglobin seems to be the rule.

It must be stated, that the opinion arrived at now, after more than a month's treatment, is that in most of the patients suffering from cutaneous tuberculosis the symptoms have been *ameliorated* but not *cured*.

One must be very careful in treating lupus with the lymph, for in the majority of cases treated here, the patients presented physical signs of tuberculosis of the lungs, when no trace of the disease could be found before the inoculation.

In surgical tuberculosis the results are variable, in some cases the symptoms are ameliorated, in others aggravated. As a rule, whenever the lesion is deeply situated swelling and pain are annoying symptoms and will require the interference of the surgeon, while if the lesion is in communication with the surface by a fistulous opening, no danger need be feared.

Injections of the lymph for laryngeal tuberculosis have been made at several hospitals, the conclusion being, that the method may be of service, but must not be used except by a specialist, who should be ready to operate if stenosis of the larynx occurs.

In a few words the opinion in Paris is that the curability of phthisis by Koch's lymph is very doubtful.

NEWS ITEMS.

State Board of Medical Education.—The following act has been presented to the Legislature of Pennsylvania by the homœopathists and eclectics:

AN ACT

To Establish a State Board of Medical Education, to Define the Powers and Duties of such Board, and to Make an Appropriation therefor.

SECTION 1. Be it enacted by the Senate and House of Representatives of the Commonwealth of Pennsylvania in General Assembly met, and it is hereby enacted by the authority of the same: That within three months after the passage of this Act, the Governor shall appoint, by and with the advice and consent of the Senate, a State Board of Medical Education, consisting of nine members, physicians in good standing, citizens of this Commonwealth, three of whom shall serve for two years, three for four years, and three for six years; and thereafter, biennially, there shall be appointed in the same manner three members of the said Board, one from each school of practice, as provided in this Section, to serve for six years in the place of those whose term shall have expired. They shall be graduates of some legally-chartered medical college, or university having the power to confer medical degrees, who shall have practised medicine or surgery for a period of not less than five years. Provided: That in the appointment of said Board the members shall be chosen equally from lists of names, each list containing the names of ten registered physicians submitted by the State Medical Societies of the Commonwealth of Pennsylvania, to the intent that the three systems of medicine—*i. e.*, homœopathic, allopathic, and eclectic—be equally represented thereon; and provided, further: That no two members shall be residents of the same county. In default of the submission of such lists, the appointments shall be made by the Governor at his discretion from among the registered physicians of the Commonwealth having the qualifications specified in this section.

Every appointment to fill a vacancy, or vacancies, in the said Board shall be for the unexpired term, and the said vacancy, or vacancies, shall be filled by the Governor within sixty days after notification of the same, in accordance with the provisions hereinbefore stated; and he shall have power to remove any member of said Board for criminal, scandalous, or dishonorable conduct.

SECT. 2. The said Board shall be known by the name and style of "The State Board of Medical Education of the Commonwealth of Pennsylvania," and shall have a common seal, and may make and adopt all necessary rules and regulations and by-laws, not inconsistent with the Constitution and laws of this Commonwealth, or of the United States, and shall have power to locate and maintain an office for the transaction of its business.

Five members of said Board shall constitute a quorum.

SECT. 3. The Board shall meet at least twice each year, at such times and places as they may determine upon after the first meeting, which shall be held within sixty days after their appointment at such time and place as the Governor may direct, at which time, and annually thereafter, they shall organize by the election from their own number of a President, and a Secretary who shall also act as Treasurer. Provided: That the President and Secretary are not of the same school of medicine.

SECT. 4. No member of the Board, except the Secretary, shall, as such, receive any salary; but the actual travelling and other expenses of any member, while engaged in the actual duties of the Board, shall be allowed and paid on presentation to, and approval by, the Auditor-General, of an itemized account with vouchers annexed.

SECT. 5. It shall be the duty of said Board of Medical Education to make all regulations as to the extent and character of the preliminary education which shall be required of all students of medicine, and notify the dean

of each medical college of the same. Any change in the standard required must be announced by the Board at least one year prior to going into effect.

SECT. 6. The Board of Medical Education shall fix the minimum curriculum and length of course of studies requisite to graduation. Provided: That the course shall not be less than four years, which shall include three annual terms of lectures. The Board shall delegate one or more of their number, who, from time to time, shall make an inspection of the methods of instruction employed, and the facilities for teaching, in each such medical college, and annually report the same to the Board.

SECT. 7. It shall be the duty of the Board to issue, under seal, to each graduate of such medical colleges as upon evidence of the dean and the secretary, or registrar of the same, shall have conformed to the requirements of the Board as herein before provided, a certificate duly setting forth such facts. Such certificate shall entitle said graduate to register in any county of the State, in accordance with the act entitled, "An Act to Provide for the Registration of all Practitioners of Medicine and Surgery," approved June 8, A D. 1881.

The fee for such certificate shall not exceed two dollars.

A similar certificate may be granted to graduates of medical colleges of other States, who may desire to practise medicine or surgery in this State, provided that such colleges stand approved by the Board of Medical Education of this Commonwealth as maintaining a standard in every respect, preliminary and final, equal to that required of the medical colleges within this Commonwealth.

The fee for such certificate shall not exceed five dollars.

Graduates of colleges having a lower standard than that required by the Board of Medical Education of this Commonwealth, may receive the certificate of the Board upon a satisfactory examination before said Board. Such examination shall be in writing, and shall apply to the preliminary education, as well as the medical knowledge of the applicant. Provided, That all questions pertaining to materia medica, and therapeutics, shall be propounded and acted upon, by members of the Board of the same school of medicine with the applicant.

The fee for each such examination shall be twenty dollars.

All examination papers shall be kept upon file, and open to inspection, for a period of two years in the office of the Board.

SECT. 8. If upon investigation the Board of Medical Education discovers, that after the passage of this act, any medical college within this Commonwealth, has granted the degree of doctor of medicine to any person, or persons, deficient in respect to either of the aforementioned preliminary or final educational requirements, or that any medical college within this Commonwealth, has conferred the degree of doctor of medicine upon any person or persons, without having passed a final examination, it shall he the duty of the Board to notify the dean of said college of such fact, and proceed against such college for such infringement of the law. The penalty for a first offence shall be a fine of not less than two hundred nor more than one thousand dollars. For a second offence, a fine of one thousand dollars shall be imposed, and the certificate of the Board of Medical Education shall be withheld from future graduates of said college, except upon examination before said Board, as provided in Section 7 of this Act. In case of conviction for a third offence, the charter of such college shall be annulled.

SECT. 9. The Board of Medical Education is hereby authorized and empowered, to bring suit for the collection of such fines, and imposition of such penalties, in the courts of the county wherein such college may be located.

All fees and fines collected by said Board shall be paid to the State Treasurer.

SECT. 10. After the first day of June, 1892, no physician shall be permitted to register in any county of this Gommonwealth except upon presentation of a certificate as provided for in Section 7 of this Act whereupon, he he or she shall be entitled, upon the payment of one dollar to be duly registered in the office of the Prothonotary of the Court of Common Pleas in the said county; and any person violating the provisions of this Act shall be guilty of a misdemeanor, and, upon conviction thereof in the Court of Quarter Sessions of the county where the offence shall have been committed shall pay a fine of not more than fifty, nor more than five hundred dollars for each offence.

SECT. 11. Nothing in this Act shall apply to commissioned medical officers of the United States army or navy or of the United States Marine-Hospital Service, nor to the member of the house or resident staff of any legally chartered medical college or university or hospital during his or her term of service therein, nor to physicians of other States meeting duly registered physicians of this State in consultation, nor to those practising dentistry exclusively, nor in any manner whatever prevent or interfere with the dispensing and sale of medicines or medical appliances by apothecaries or pharmacists, and nothing in this Act shall be construed to prohibit the practice of medicine and surgery within this Commonwealth by any practitioner who shall have been duly registered before the first day of February, 1892, according to the terms of this Act, entitled "An Act to provide for the Registration of all Practitioners in Medicine and Surgery," avproved the 8th of June, Anno Domini, 1881.

SECT. 12. The Board shall make to the Governor, on or before the first day of December of each year, a full report of the work of the Board for the year.

SECT. 13. For the purposes of this Act, the words "practice of medicine or surgery," shall mean to treat, or attend, any person for money, gift, or reward.

SECT. 14. The sum of two thousand dollars is hereby appropriated to meet the necessary and legitimate expenses of the said Board.

SECT. 15. Section 4 of an Act entitled "An Act to provide for the Registration of all practitioners of Medicine and Surgery," approved the eighth day of June, Anno Domini 1881, is hereby repealed.

Obituary.—Edward Bellamy, F.R.C.S., senior surgeon to Charing Cross Hospital, London, died January 4, 1891, of acute pneumonia, at the age of forty-eight years.